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# SECTION ON

# OPHTHALMOLOGY

OF THE

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# TO THE MEMBERS OF THE SECTION ON OPHTHALMOLOGY

### OF THE

# AMERICAN MEDICAL ASSOCIATION

All the completed papers for the program of the Section on Ophthalmology of the American Medical Association, Atlantic City Session, 1914, will be found herein. Provision has been made for notes on the margins of the pages and on the blank page following each paper.

Following the plan in force during the last few years, the papers will not be read before the Section, but each author will be allowed not to exceed ten minutes in which to summarize the points in his paper and to introduce discussion. The member appointed to open the discussion of any paper will be allowed not to exceed ten minutes and subsequent speakers five minutes. Any one who has written out his discussion will of course hand it to the stenographer. (It should, of course, be remembered that written discussions must not exceed the limits of length imposed by the time allowed to each discusser; that is, about 1,000 words for those who open discussions, and about 500 words for those who follow. Discussers will naturally prefer to see themselves that their discussions conform to these requirements, rather than run the risk of having their remarks condensed by other hands.)

Attention is called to the desire on the part of the officers of the Section to have an exhibition of pathologic specimens, photographs, charts and printed histories of rare and interesting cases. Members are also invited to exhibit new instruments and appliances which may be of interest to the members of the Section. Those desiring to contribute to this feature are requested to correspond with the Secretary of the Section.

George S. Derby, Secretary.

Frank C. Todd, Chairman.

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# TUMORS OF THE OPTIC NERVE *

W. GORDON M. BYERS, M.D.
MONTREAL

This paper is intended as a contribution to the surgery of those primary tumors which occur in the optic nerve. As long ago as 1901, from the laboratories of the Royal Victoria Hospital, I issued a monograph, with which possibly some of you may be acquainted, dealing more especially with the pathologic aspect of the subject. I now propose to lay before this Section of the American Medical Association the experience which I have acquired since that time in the clinical aspects of these cases and in the operative procedures for their removal. As preliminary to that, however, I shall be obliged to define more clearly the conclusions at which I have arrived on the pathology of these growths.

Simultaneously with the arrival of the invitation that I should address this meeting, I encountered a clinical experience which encouraged me to undertake the task, for tumors of the optic nerve are of such rare occurrence that most men, even in a life's experience, do not encounter them; and a surgeon seeing the condition for the first time must almost of necessity turn to the literature for guidance.

Let me, then, direct your attention to a few facts concerning the pathology of these growths. In the investigations spoken of, I came to the conclusion, with very material assistance from Professor Adami, in whose laboratory at the Royal Victoria Hospital, Montreal, my studies were conducted, that tumors of the optic nerve, growing within its dural sheath, could be rather sharply divided into two main groups. In the first place one had to do with growths the basis of which was essentially an overgrowth of the

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fibrous connective-tissue elements of the nerve, but very protean in character, inasmuch as one found every phase of development from the embryonic myxomatous type to firm fibrous bands. The appearances were unquestionably influenced by conditions of stasis in the lymph and venous channels which resulted solely from the local anatomic arrangement of the parts; and altogether the changes suggested those characteristically present in cases of elephantiasis. These findings were in agreement with those of practically every student of the subject as a whole, and can still perhaps be best described by the term "fibromatosis," which I used in the title of my first contribution to the subject.

In the second group of neoplasms the changes, varying somewhat in character, were attributable to a proliferation of the local endothelial cells, and the growths were classified as endotheliomas.

It seemed, too, from our studies, that these two groups were histogenetically closely related, that even, at times, it was possible for the indeterminate fibrous tumors to take on the more striking arrangement of the endotheliomas. As regards frequency I found from an analysis of the entire literature on the subject that the fibrous growths outnumbered the endotheliomas in the proportion of about thirty-five of the former to one of the latter.

Unfortunately, we know so little about the final outcome of these cases that it is impossible to make very positive statements in regard to their clinical behavior. We know that they are characteristically slow in their development; and the reports seem to show that recurrences, always of a purely local character, are rare, secondary growths being perhaps commoner after the endotheliomas than after the fibrous tissue-group. In this connection let me say again, however, what I previously laid great stress on in speaking of the prognosis in these cases, that in many, if not in the majority, of these cases, the tumor of the optic nerve is but part of a neoplasm with a more or less wide intracranial distribution which in its eventual development will bring about the death of the patient. Whether or not it is possible and permissible to do anything further in those cases in which it is perfectly clear after the removal of the orbital growth that an intracranial portion remains, is still an open question.

In considering this subject it is of equal importance to make note of the primary extradural tumors of the optic nerve, that is, growths which take their origin in, or from the outer surface of, the dural coat of the nerve. While by no means so common as the intradural growths, occurring probably in the ratio of about 1 to 5 or 6, they are more frequently encountered than the intradural endotheliomas whose histologic structure they almost invariably possess. Parsons' careful analysis of the published cases led him to believe that almost certainly at least three-fourths of these growths were of this nature, the remaining tumors being of a frankly sarcomatous type. Without entering into a consideration of the possible relationship of these tumors to the intradural variety, or attempting any conjecture as to their origin, one can say that, while these growths are, too, like the intradural variety, characteristically "slow and even" in their development, they are yet more rapid in their growth than the intradural type, as one would expect from the absence of the retaining dural sheath; that their removal is more often followed by recurrences, and that they may even, if Lawson's case be admitted (though it is questioned by Parsons) be followed by metastases. In a word, the extradural tumors are distinctly of a more menacing type than those within the dural sheath.

The fact that most if not all of the intradural connective-tissue tumors are of a non-malignant character, and that these greatly outnumber the endotheliomas, might incline one to adopt an "expectant" attitude, especially in those cases in which, as often happens, the neoplasm over long periods seems to be making little or no progress. This is especially true when other conditions are present that favor a policy of waiting, that is, pregnancy, the need of nursing a child, or profound nervous or other systemic disturbance; and always there are to be considered the dangers inherent in the operation and in the anesthetic, and the question of mutilation, especially in women.

A careful consideration of this attitude leads one, however, inevitably to the conclusion that it is incorrect from every point of view. In the first place, because it is impossible to differentiate clinically the three types of growths whose pathologic features have just been described. All these tumors have exophthalmos, in or near the line of the orbital axis, and profound or complete loss of sight as their characteristic symptoms; but, while it can be said that the possibility of an intradural tumor diminishes and that of an extradural neoplasm rises in proportion as the degree of sight increases, this statement can be taken only very generally; for there are marked exceptions on both sides. The ophthalmoscopic changes and the other signs as described are, too, of no value in making a differential diagnosis; and even the age factor coincides. A surgeon can never be certain, then, that he has to do with a growth of the "fibromatosis" type; he has always to keep in mind the possibility of an intradural endothelioma or more especially of an extradural neoplasm of this nature. An attitude of expectancy here can only give rise to conditions which at best will necessitate a more radical operation, and at worst jeopardize the patient's chances of recovery.

In the second place, a waiting policy is unjustifiable in tumors of the optic nerve because one cannot ever be sure of the size, situation, or rate of development in any given growth, at any given moment. It is notorious that in these cases the exophthalmos, the principal index, is often pronounced out of all proportion to the size of the neoplasm, and that it frequently undergoes variations that bear no relationship whatever to the dimensions of the swelling. The explanation of these features is to be found in conditions of stasis and edema in the lymph-spaces in and about the optic nerve and eyeball; and perhaps at times in the buckling of a piece of uninvolved nerve. There are so many exceptions that it can be said only in a very general way that early pain, conspicuously absent in these cases, would rather suggest a development in the apex of the orbit, and an early neuritis or atrophy involvement of the anterior portion of the nerve. Increasing hypermetropia, valuable when present, too often fails us to be relied on; and palpation is seldom of any assistance, and may even be misleading.

Finally, considering the matter from the point of view of the operator who wishes while removing the growth to retain the eyeball *in situ* (and the desirability of this effort is now so well established that I shall not take up your time in defending the position), there is every reason in favor of early and immediate interference. It can be affirmed with emphasis that no matter what procedure is adopted, the larger the growth the greater the manipulation that is necessary, with its consequent dangers to the cornea and to the blood-supply of the globe, and the fewer the chances of achieving the end desired. In illustration of the points which I have tried to adduce let me cite the two following cases:

CASE 1.—Miss B., a nurse, aged 44, consulted me on Jan. 25, 1910, for blurring of the right eye noticed during the preceding few weeks. The ophthalmoscope showed no changes in either eye, but, while the vision of the right side equaled 6/5 —, that of the left was only fingers at 1 foot. The diagnosis, uncertain at first, became, five months later, clearly one of tumor of the optic nerve, when slight proptosis appeared along with beginning pallor of the optic disk.

The patient had a most deeply rooted objection to an operation and dreaded the disfigurement. Influenced by her and by an imperfect apprehension of the factors which I have already set forth at length, I decided to follow the case in an expectant way.

The proptosis developed very gradually during the next three years without any change in the refraction of the eyeball and with only slight involvement of the musculature; but during the summer of 1913, the rate of progress was much more rapid, the exophthalmos becoming noticeably greater, the excursions of the globe much more restricted, and severe neuralgic pains a pronounced feature. The patient's general condition was somewhat impaired, apparently, however, from pain and loss of sleep rather than from the direct influence of the tumor on her constitution.

On the patient's admission to the hospital, Oct. 15, 1913, the eyeball showed a proptosis, directly in the line of the axis of the orbit, of about 2 cm.; total restriction of movement, except downward and inward, where the excursion was about half the normal; complete atrophy of the optic disk; and nothing on palpation. No changes were made out in any other part of the body; and the other eye was quite normal in every respect. The growth was removed October 16, in the manner described later on, and recovery was quite uneventful. At present there is no sign of recurrence, and the patient's general condition is excellent.

The macroscopic examination of the orbital contents showed a large tumor-mass surrounding the posterior surface of the globe and extending into the orbital tissues beyond the muscular cone; but the dural sheath was so disintegrated and invaded by the growth that it was impossible to say whether the neoplasm had originated within or without this structure. Microscopic examination also failed to clear up this point. The growth was pronounced to be "fundamentally endotheliomatous" by my colleagues, Drs. Gruner and McMillan, who sent me the following minute description: "The tumor consists of lobulated masses of triangular shape, separated by broad connective-tissue septa. The lobules are partly solid and partly take the form of acinous tissue, with numerous polygonal spaces of fairly uniform size, filled with material which stains blue with hematoxylin and pink with carbolthionin. The adjoining spaces are separated by two layers of cells, the lining of each space, respectively. There appears to be no fibrous tissue between the acini at all, and, speaking broadly, many fields can be found which closely resemble normal thyroid tissue. The angles between the spaces are filled up with cells similar to those lining them, and thus solidity is imparted to the mass. The cells lining the spaces are small, cubical or slightly flattened, and have a relatively large, intensely staining nucleus of oval shape; but definite cell-borders cannot be distinguished. The cytoplasm is rather acidophil; occasionally, a few cells are found mixed up with the mucoid matter of the space. Some of the cells show a tendency to vacuolization in the vicinity of the nucleus; but the cytoplasm does not develop a foamy structure, however abundant the mucoid material may be. The shape of the lobule is not always the same; a number of them are elongated while others differ in having no definite outline and being made up of ramifying, linear groups of cells devoid of any lumen formation. Transition forms between this type and the first described are to be found. These are areas in which the material separating the ramifying column is undergoing mucinous change, staining purple instead of pink or blue, but otherwise showing the same appearance as is seen in the gland spaces already referred to. Another type of lobule is that composed of densely packed, darkly staining, small rounded cells without any lumen formation of any kind. The stroma of the tissue is composed of very coarse fibrous tissue in which there are few cells. It is variable in thickness, the smallest lobules being enclosed by the largest amount. In many cases the septa become converted into hyaline lines staining brightly with eosin. The muscular bundles are pushed aside by those nodules of growth which are present in that situation."

The history of my second case is a curious story of functional disturbance and of simulation based apparently on numerous organic troubles. What relationship all these bear to the changes in the optic nerve it is of course impossible to say.

Case 2.—The patient first came under observation at the Royal Victoria Hospital for profound chlorosis and gastric ulcer. Three years later she was operated on at the same institution for appendicitis; and shortly after this, Oct. 5, 1900, I saw her in the eye clinic for headaches which were worse after reading and sewing and had been especially bad since her abdominal operation. On the occasion of her first visit her sight was 6/6 on both sides, with a manifest hypermetropia of half a diopter; and the fundi were unquestionably normal. Following the instillation of a mydriatic, she developed within the next few days the most typical example of functional amblyopia I have ever seen, great reduction of the central vision with contraction and inversion of the visual fields. She had typhoid fever in December, 1906, with a miscarriage during the course of the attack, and, following this, functional hemianesthesia. A year later, Feb. 15, 1907, she again came under my observation in the eye clinic, and I was surprised to find unmistakable signs of a tumor of the right optic nerve, and slight proptosis, with complete atrophy of the optic disk, the other eye being normal.

During the following years the patient had repeated miscarriages attributable to uncontrollable vomiting that her pregnant state engendered, and several gynecologic operations for the relief of uterine disturbances. I saw her a few times during this period, but her wretched state of health, coupled with the fact that the tumor showed little or no apparent progress, seemed to justify a waiting policy. Following a severe operation on one of her kidneys in the spring of 1913, the condition became, however, rapidly worse; and when I saw her in the autumn of the same year and advised removal of the tumor she consented to have it done.

At the time of admission to the Royal Victoria Hospital, Jan. 19, 1914, little or no restriction of the ocular excursions accompanied the moderately marked proptosis, and nothing could be made out on palpation. The ophthalmoscopic picture was that of a secondary atrophy of the optic nerve with the deposition of a good deal of fibrous tissue on the disk and along the vessels below. There was also an irregular band of atrophic retinitis running horizontally through the macular area.

The following day the tumor was removed in the manner which I shall presently describe, the eyeball being left in position. For several days following the operation there was a marked milkiness of the cornea suggesting the appearance which one sees following a lime burn of this structure; and there was marked pericorneal congestion. There remains now an apparently permanent opacity of the lower half of the cornea with superficial vascularization as in pannus, and this, together with a marked layer of exudation in the pupillary area, prevents one from obtaining anything but a dim red reflex from the fundus. The iris still remains somewhat discolored, the globe is slightly shrunken and squared, and the tension is quite soft; but the movements of the eyeball are practically full, and, though the lids are somewhat retracted, the general appearance is good.

The excised tumor measured 3 cm. in length by 1.75 cm. at its point of greatest breadth. It strongly suggested a phimotic penis in appearance, owing to the fact that it tapered markedly toward its posterior end and was constricted about its middle by a band of fibrous tissue. A macroscopic examination showed that the removal of the tumor at the posterior pole of the globe was incomplete, as was also the eradication behind. Microscopically the growth belonged unquestionably to the group of connective-tissue overgrowths which I have described at the commencement of this paper and very fully dealt with in my previous monograph.

The operations performed in these two cases, in both of which the principle of saving the eyeball while removing the growth was followed, can be described briefly.

In my first patient I employed Kroenlein's procedure, which consists I may say briefly for those not familiar with its details, in removing the tumor through an opening made in the external wall of the orbit. The only variation from the operation as originally described was that I used a strong septal saw to make the bony incisions instead of the chisel and mallet as recommended by Kroenlein. On entering the orbit I encountered a soft, almost gelatinous tumor, much larger than I anticipated; and it soon became apparent that it had grown to such a size and had infiltrated the tissues to such an extent that it was impossible to remove it through the lateral aperture. Accordingly a complete evisceration of the orbit was carried out after the osseous resection was replaced and the overlying flaps closed. Recovery was quite uneventful, the removal of the orbital contents not retarding in any way the healing of the incisions made during the first operation. At present there is no sign of recurrence, and the patient's general condition is excellent.

In view of the difficulties I had had with my first patient, I decided to employ a modification of Knapp's procedure rather than the Kroenlein operation to remove the tumor in my second case, because it was apparent that in this instance, too, the neoplasm had attained a considerable size. After tenotomizing the external rectus and opening the conjunctiva and Tenon's capsule for a short distance above and below, I had no difficulty in separating the sharply defined intradural growth from the surrounding structures; and I succeeded in removing the neoplasm very completely, and with practically no hemorrhage, by means of a stout tonsil snare, which lent itself very nicely, it seemed to me, to this purpose. Following this the external rectus was reattached and the conjunctival opening closed by sutures.

Criticizing Kroenlein's operation and Knapp's procedure in the light of this recent experience, and of previous cases in which I have made use of the latter method, I would say at the outset that Kroenlein's operation is adapted to the removal only of small, or at most, medium-sized growths. At best, one feels hampered by the smallness of the lateral opening; the deeper parts of the orbit are not so readily seen as one would think, owing to the tendency of the contents of the cavity to prolapse into the wound; and it is actually impossible to deliver large growths, except piecemeal, through the biggest aperture that can be made. On the other hand, the vascular supply of the eyeball is least interfered with by this method; the cornea perhaps is not quite so exposed to injury as in Knapp's procedure, and the orbit is not opened to infection from the conjunctival sac.

The Knapp method is suitable for any sized tumor of the optic nerve, it being possible with external canthotomy for an operator to procure practically any sized aperture he wishes. The only objection that can be made to it is that it opens up the orbit to infection from a field which cannot be sterilized. In the absence of tear-sac trouble, and with bacteriologic control, this fear, however, can be disregarded. With the method one works easily in an anteroposterior direction; and in those cases in which the tumor is not infiltrating the orbital tissues, these structures are prevented from prolapsing into the field of operation by Tenon's capsule. Further, with this procedure, if the operator finds the growth ineradicable, he can proceed to evisceration of the orbit without loss of time, and without the injury to the adjacent parts incidental to Kroenlein's operation. Viewing the matter as a whole, I look, then, on Knapp's method as easier to perform and more suitable for these cases than Kroenlein's procedure; and I am convinced that it alone can be satisfactorily employed when the growth has attained large dimensions.

The two cases which I have cited brought with them an experience which materially altered my views in regard to the treatment of tumors of the optic nerve.

My previous work had obscured the potentialities of the few reported cases of intradural endothelioma, and I had not fully grasped the more menacing characters of the extradural tumors, which had not come within the scope of my paper. There remained only the impression of the connective-tissue growths, little if at all malignant, and characteristically slow in development; and the feeling that these could be followed in an expectant way, until pain or some such indication demanded their removal, much in the same manner as one does a fibroma of the uterus. My conviction in this matter was strengthened by the emphasis laid on the benignancy of these growths by numerous other investigators whose findings were substantially in accordance with my own; but it is clear now that the contingencies which I have mentioned have not been clearly apprehended or sufficiently accentuated, because the general conceptions of tumors of the optic nerve have been based almost exclusively on studies of the intradural connective-tissue variety.

The expectant attitude adopted in my second case, largely as the result of conditions which could not be controlled, was to a certain extent justified by the finding of a tumor of the benign fibromatosis type and by the ease with which it was removed. The changes in the intracranial portions of the nerve, which the microscopic examination clearly showed, occurred in all probability concomitantly with those in the orbital division. This supposition is based on the post-mortem findings in several cases of this kind to which I have already referred; but it is just possible that one had to do here with one of the more uncommon occurrences in which the growth develops in the anterior end of the nerve, leaving for a varying length of time a piece of uninvolved nerve between the tumor and the optic foramen; and that earlier interference might have resulted in an entire eradication of the process. In my first patient, however, it is apparent that under the same clinical picture one had to do with a growth of an unquestionably more malignant nature, as shown by its histologic character and its behavior in the tissues; and though I am persuaded that the tumor involved the cerebral end of the nerve from the outset, and know that the bony walls were not invaded, there is no question now in my mind that the right thing to have done in this instance was to remove the growth as soon as the nature of the condition became clear. Further, in view of the occurrence of neoplasms of this sort, I feel that the only safe practice to adopt in tumors of the optic nerve is to extirpate them as soon as possible after the diagnosis has been made.

346 Mountain Street.

# MEMORANDA

# OCULAR MANIFESTATIONS OF THE TOXEMIA OF PREGNANCY

## CLINICAL REPORT OF A CASE *

WARD A. HOLDEN, M.D.
NEW YORK

We know little about the ocular manifestations of eclampsia and their causation, and this single case is put on record because it seems to throw a little light on this obscure subject.

### REPORT OF CASE

*Patient.*—Feb. 6, 1913, I was called to the Sloane Hospital for Women to examine the eyes of Mrs. G., housewife, aged 25. The following notes as to her condition while in the hospital are taken from the hospital records by the kind permission of Dr. E. B. Cragin and Dr. E. C. Lyons, the resident obstetrician. Feb. 3, 1913, the patient was admitted to the hospital and said that she was about seven months pregnant, for the second time; that she had vomited during the first three months of her pregnancy, and that for a month her feet had been swollen at night. Her chief complaints, however, were that for five days she had seen double, and that during this time the sight of each eye had grown rapidly worse. Her general condition seemed to be good, there was no toxic rash, the legs and feet were slightly edematous, the blood-pressure was 160 mm. and the urine (51 ounces on February 4) contained 20 per cent. albumin and many cells, and was alkaline. She was kept in bed on active eliminative treatment, and the urine increased greatly in volume and the percentage of albumin was reduced, while the reaction became acid.

*Examination of the Eyes.*—February 6 I found a paresis of the left external rectus and an absolute central scotoma in the field of each eye so extensive that vision was reduced to the recognition of movements of the hand, but this was possible in the entire periphery of each field. The fundi were normal in appearance except for a trace of retinal edema about the macula lutea in each eye, which was not sufficiently marked to cause obscuration of vision.

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*Course.*—I advised the immediate induction of labor in order to save her vision, and the patient was at once delivered of an eight-months still-born child. From an obstetric point of view, she had a rapid and uncomplicated recovery, and her sight became rapidly better, while the albumin was reduced and the blood-pressure fell and she was discharged in good condition February 22.

March 3, 1913, I saw the patient again and found that her vision had returned within a few days after the operation and was 20/20 in each eye with normal fields. There had been complete recovery from the paresis of the external rectus also; but she said that since leaving the hospital she had experienced much difficulty in seeing at night; and in each macular region of the retina, which had, a month before, been edematous, there was now an abnormal pigmentation. The fundi were otherwise normal.

July 22, 1913, the night blindness was said to be less pronounced, but the abnormal pigmentation was found to have spread from the macular region out to the periphery of each retina. On careful examination it was seen that the pigment of the retinal pigment epithelium had become rarefied in punctate spots and greatly increased in adjacent punctate spots. In the macular region many of the accumulations of pigment were confluent, giving this region a much darker appearance than normal when viewed with the ophthalmoscope by the indirect method. No colloid excrescences were found in the retina and the pigment seemed not to have wandered into the retina proper. The vision was 20/20 in each eye. At this time there was still albumin in the urine and the patient complained of vertigo, pain in the back and transitory obscurations of vision. The Wassermann blood reaction was negative.

Oct. 9, 1913, the pigmentary changes were more pronounced.

Jan. 29, 1914, the condition was unchanged.

This curious chronic progressive disturbance in the pigment epithelium of the retina would seem to be due to the same toxic condition of the blood which, when more intense, caused paresis of one external rectus muscle and the retrobulbar affection of the optic nerves. The night blindness of scurvy, for example, must be attributed similarly to the action of the altered blood on the pigment epithelium, and this point will be mentioned later.

In the absence of an anatomic examination we can merely conjecture what was the cause of the paresis of one external rectus and of the large central scotoma in the field of each eye with normal optic disks and speedy recovery after delivery.

Weil and Wilhelm,[1] recent investigators of ocular disturbances in pregnant women, believe that the retention in the blood of certain salts causes hydropic disturbances—edemas, and eclampsia, with sudden blindness without ocular changes; while the retention of nitrogenous substances causes albuminuric retinitis and renders the prognosis grave.

The characteristic lesion in eclampsia is a more or less localized edema of the intracranial tissues, of sudden onset. Complete transitory blindness with normal fundi is a fairly frequent symptom of eclampsia. In the rare cases of incomplete recovery of vision, a concentric contraction of the fields down to 10 degrees in each meridian, with good central vision, has been found. Lagrange and Abadie each has reported such cases, and Endelmann, describing a similar case, correctly called it double homonymous hemianopsia with preservation of a small central field in each eye. A homonymous hemianopsia on one side, followed later by a homonymous hemianopsia on the other, with preservation of a small central field in each eye, has been observed repeatedly in patients with advanced arteriosclerosis, the bilateral cortical or subcortical hemorrhages or thromboses sparing some of the fibers serving central vision.

In many of the cases of blindness in eclampsia we must suppose that there is an edema affecting the calcarine cortex or the optic radiation on each side, which in some cases causes destruction of tissues and therefore produces permanent defects in the fields of vision.

The question arises whether in our case also the visual and sixth-nerve symptoms might not have been caused by a bilateral edema of some parts situated near the optic chiasm, for it is not likely that central scotomas clearing up in a few days, with disks and retinal vessels normal at the time and also months later, were due to a true inflammation of the optic nerves.

Leber,[2] nearly forty years ago, offered the suggestion, apparently overlooked by most later writers, that a retrobulbar neuritis coming on suddenly after a coryza, with slight fundus changes, might be due to a periostitis of the optic foramen—a suggestion that is fully confirmed by Onodi in the recent elaborated English edition of his "Optic Nerve and Accessory Sinuses." Onodi finds that in the cases of retrobulbar neuritis due to sinusitis, there is a direct transmission of the infection from the lining membrane of the sinus to the lining membrane of the optic foramen, either through a dehiscence in the thin bony wall or along the perforating veins. The swollen periosteum compresses the optic nerve, affecting first the delicate axial or papillomacular bundle of fibers of the optic nerve, and thus causes a central scotoma.

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[1] Weil and Wilhelm: Obstétrique, iv, No. 3.

[2] Leber: In Graefe-Saemisch Handbuch der gesamten Augenheilkunde, Ed. 1, 5.2, p. 814.

Foster Kennedy[3] has put on record half a dozen cases of tumor or abscess of one frontal lobe of the brain, producing a central scotoma in the field of the eye on the same side by a direct pressure on the optic nerve behind its entrance into the optic foramen. I have observed two cases of this kind. Furthermore, though some others have not had the same experience, I found that a tumor surrounding the sheaths of the optic nerve far back in the orbit caused a sharply defined atrophy of the axial bundle of fibers alone.

The conjecture, therefore, would be that an edema —possibly of the anterior lobes of the brain, but more likely of the meninges and of the periosteum of the optic foramina which is continuous with the dura mater—brought about pressure on each optic nerve near its origin and on one sixth nerve which, as it passes to the sphenoidal fissure, lies very near the optic nerve.

The night blindness in this case was preceded by edema of the retina and of the ankles in a patient with supposed retention in the blood of saline elements. In scurvy, some of the saline constituents of the blood have been found to be in excess. Hence, it may be that, apart from the sclerosis of the choroidal vessels that leads to the degeneration of the pigment epithelium and thus causes night blindness in retinitis pigmentosa and other choroidoretinal affections, an excess of certain salines in the blood may be the cause of the faulty functional activity of the pigment epithelium that produces night blindness of a transitory character.

8 East Fifty-Fourth Street.

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[3] Kennedy, Foster: Am. Jour. Med. Sc., September, 1911, p. 355.

## 27

# MEMORANDA

## MEMORANDA

# TWO CASES OF ACUTE RETROBULBAR NEURITIS, ASSOCIATED WITH MARKED ACETONURIA*

LEE MASTEN FRANCIS, Ph.B., M.D.
BUFFALO

The two cases of acute retrobulbar neuritis presented in this report are of interest because a thorough physical examination by competent observers revealed no definite findings except the presence of a pronounced reaction for acetone in the urine. Both patients were young, one a girl of 8½ and the other a man of 31. The girl had been an exceptionally healthy child, while the man gave a history of a previous attack of colitis coincident with marked acetonuria. Both lived under ideal hygienic conditions, being members of families in more than comfortable circumstances. In both cases, hysteria, disease of the nose and accessory air-passages, alcohol, tobacco and drugs could be eliminated with certainty, nor were there any grounds on which the likelihood of chemical or ptomain poisoning might be entertained.

While blindness from retrobulbar neuritis occurs in diabetes, as a rule the patients are past middle life, very much broken down, or well advanced in the disease. I have not been able to find cases reported occurring in young persons associated with a decided acetonuria and with no other likely etiologic factor.

Case 1.—A. A., aged 8½, the daughter of well-to-do parents, consulted me Feb. 12, 1910. The family history was good. The child had always been exceptionally well and healthy and had lived under ideal conditions. There was no history of gastric disturbances, no coryza, injuries or illnesses of any sort. No visual disturbances had been observed by patient, parents or teachers, until a few days ago the girl complained that she could not see the blackboard in school and that the print in her books was not clear. At about the same time she complained of dull frontal headaches, and her mother remembers that she was somewhat nauseated when riding on the street-cars.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

*Examination.*—The patient is a well-nourished, healthy appearing girl of normal size and weight, of good color and presence. The pupils are of normal size, equal and respond readily to all reflexes.

O. D. V. = 6/15
O. S. V. = 6/15

Muscles: 1° exophoria with Maddox rod. The fundus examination without mydriasis showed the disks to be of normal appearance, retinal vessels normal. Not suspecting the nature of the case, I ordered a 1 per cent. atropin solution to be instilled three times daily, and the patient was requested to return three days later for refractive examination under cycloplegia.

February 15, the patient returned, the atropin having been used as ordered.

The retinoscope showed:

O. D. + 1.00 cy. ax. 105°
O. S. + 1.00 cy. ax. 75°

Vision (under atropin):

O. D. V. = 6/22.3 + 1
O. S. V. = 6/22.3 + 1

A careful test with trial lenses showed that while the cylinders indicated by the retinoscope made the test-letters more distinct, they did not improve her ability to read. A test was then made of her visual fields by means of colored disks on the end of a rod. The fields for white appeared normal, but there was a total lack of recognition of the red disk in the fields of fixation. The blue field appeared normal; green and yellow were not tested.

The eyeballs were not tender on pressure. The fundus examination revealed no change in the appearance of the nerve-head or retinal vessels.

The patient was then referred to Dr. F. W. Hinkel for examination of the nose and air-passages and to Dr. N. G. Russell for a thorough physical examination.

The examination of the nose and air-passages was reported as absolutely negative.

Dr. Russell's report is as follows: I have examined A. A. carefully and have found her normal in every way except possibly somewhat nervous. The blood is fully up to standard except that hemoglobin is a little low. The urine is perfectly normal except a decided reaction for acetone and a slight reaction for diacetic acid. This is rather unusual in a full diet such as she has been on. ....

Feb. 21, 1910, the patient returned to me, the effects of the atropin having thoroughly passed away. At that time:

O. D. V. = 6/12 + 1
O. S. V. = 6/12 + 2

Plus cylinders, while improving the above letters do not add to capacity to read smaller letters. The fields were then charted on the perimeter (Fig. 1.).

A urinalysis made by Dr. Russell, February 25, showed a mild acetone reaction but no diacetic acid. Having been under a regulated diet, ordered by Dr. Russell, the patient returned to me, April 25, 1910, at which time O. D. V.=6/6, and O. S. V.=6/6. The urine has been free from acetone about two weeks. A test with a red disk showed no color defects. The patient again came under my observation in December, 1910, when she was brought to me complaining of asthenopia and headache dependent on eye use. Her visual tests at that time were as follows:

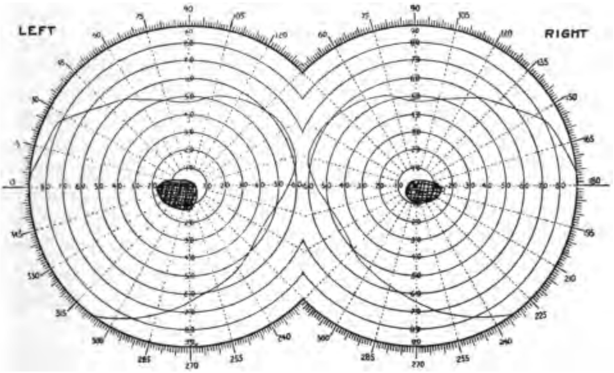

Fig. 1.—Absolute scotoma for red, white and blue fields normal.

O. D. V. = 6/9 — Exophoria 1½, fields negative.
O. S. V. = 6/9 + Fundus negative.

These lenses were ordered and worn during school hours and for near use with a complete relief of the symptoms.

May 28, 1913, the patient again returned, saying that the lenses had not been worn during the past year. For the past two weeks her headaches and asthenopia had returned. Visual examination revealed:

O. D. V. = 6/9 — Exophoria 1½, fields negative.
O. S. V. = 6/9 + Fundus negative.

Under atropin:

O. D. V. = 6/75 — 0.25 sp. = + 0.50 cy. ax. 90° V. = 6/5
O. S. V. = 6/6 + 0.50 cy. ax. 75° V. = 6/5

A full correction was ordered for school use, which has been worn with comfort.

CASE 2.—*History.*—A. G., aged 31, married, department manager of a business firm, consulted me, Jan. 22, 1911. Family history is without incident. He uses neither tobacco nor alcohol, denies venereal disease and describes himself as of "nervous disposition" and as having a "sensitive stomach" and "somewhat run down" from overwork. He says that for many years he has had occasional attacks of what he describes as "intestinal catarrh," for which his physician has prescribed exercise and diet, with relief of symptoms. He has worn lenses for many years, having been examined by Dr. Julius Pohlman of Buffalo. He is wearing:

O. D. + 0.75 sp. = — 2.00 cy. ax. 180°
O. S. + 0.50 sp., = — 1.00 cy. ax. 180°

The glasses have always been comfortable and have given him good vision.

*Present History.*—He has been in his usual good health so far as he knows, except that he remembers having had a slight attack of indigestion a few days ago which he ascribes to a corned-beef sandwich which he ate at noon. January 19, he noticed that the vision of his left eye was blurred and the sight noticeably impaired. The vision has remained about the same as at first, so far as he can determine. There have been no headaches or dizziness or "colds in the head." His work is not that which would expose him to any poisons in its performance, and he has been taking no medicines of any kind.

*Examination.*—The patient is somewhat pale, is fairly well nourished and of apparently high-strung disposition. Pupils are of normal size and equal, and respond readily to light, convergence and consensually. There is no tenderness on firm pressure on the globes.

O. D. V. (tested with his lenses) = 6/6
O. S. V. (tested with his lenses) = 6/15
Muscles = 2° exophoria (Maddox rod).

The ophthalmoscopic examination shows the disks to be sharply outlined, of normal color and appearance. The retinal veins and arteries are normal. The circulatory balance is good. There are no abnormal appearances to be noted.

The fields were then tested by means of disks on a rod. While the visual field of the right eye appeared normal for white, blue and red, on the left side white and blue were normal. The patient fails to recognize the red disk in the center of the field. The perimetric chart was then made (Fig. 2).

A diagnosis of left retrobulbar neuritis was made. Dr. W. Scott Renner, Buffalo, who made the rhinologic examination, reported that the nose and sinuses were free from disease. Dr. Herbert M. Hill reported a twenty-four-hour sample of urine negative, both chemically and microscopically, but no test for acetone was made. A competent internist, to whom the patient was referred in the temporary absence of his regular medical adviser, reported to me within a few days that he had been unable to discover evidences of hysteria or anything abnormal about the patient. The differential blood-count was negative. Hemoglobin estimate was 75 per cent. At my suggestion the patient was given small doses of calomel, sodium salicylate and sweat-baths. The following days were without incident, beyond the fact that the patient was very apprehensive concerning his condition and became very nervous. I suggested a rest and change of scene. The patient decided that he wanted to go to Lakewood, N. J., so I gave him a letter to Dr. Arnold Knapp of New York, instructing him to consult him during his absence. A few hours before leaving for Lakewood, however, his regular physician, Dr. John Mesmer, having returned to the city, I suggested that he submit a sample of urine to him, and seek his advice. This was done just before the patient's departure.

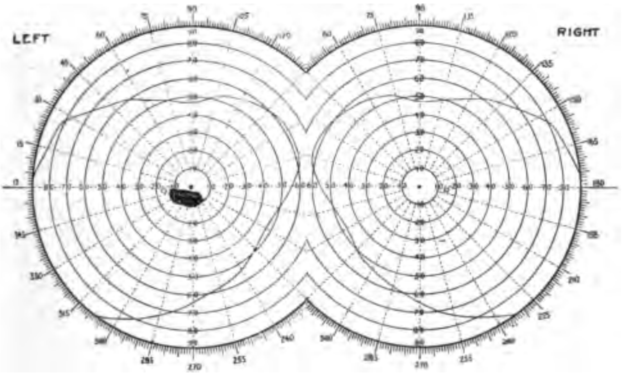

Fig. 2.—Absolute scotoma for red, relative for blue, white normal.

Within a few days Dr. Mesmer reported to me that the examination of the single sample of urine submitted to him had shown "a very heavy acetone reaction." He also reported that the patient in past years had had several severe attacks of "mucous colitis with crises" and in each instance there had been decided acetone reactions. Each attack had yielded readily to a proper diet and outdoor life with plenty of exercise. During the intervals between the attacks the urine had been free from acetone bodies. Dr. Mesmer had advised the patient to return to his dietary regulations, and to take plenty of exercise during his sojourn at Lakewood.

Following are the clinical records of the case tested with lenses:

Feb. 16, 1911: O. S. V. = 6/7.5 + 3 Scotoma for red tested roughly appears smaller.
Feb. 27, 1911: O. S. V. = 6/6 + 3 Urine is free from acetone.
Mar. 29, 1911: O. S. V. = 6/.6 + 2
Apr. 22, 1911: O. S. V. = 6/6 + 2 No scotoma for red.

April 22, 1911, the patient's refraction was gone over under homatropin with the following result:

O. D.—0.62 sp. = + 2.25 cy. ax. 90° V = 6/5
O. S. + 0.62 cy. ax. 90° V = 6/5

The urine gives no acetone reaction. During the past three years the patient has been seen at frequent intervals. During this time visual acuity has remained the same. There have been no attacks of lowered vision in the right eye.

According to von Noorden,[1] "the primary acetone bodies are beta-oxybutyric acid and aceto acid, from which acetone is probably derived as a secondary product . . . by far the most important sources of acetone are the lower fatty acids." Whatever be the source or whether the virulence of the toxin in cases of faulty metabolism be acetone or some antecedent product or products, clinicians recognize that the excretion of acetone is a fair index of a toxemia. While acetonuria is not at all infrequent in children, and to a less extent in adults with gastro-intestinal disturbances, and invariably accompanies starvation, it may be fairly stated that acetone is not found in appreciable quantities in the urine of normal persons, and that its presence is an index of some toxemia of enough virulence to attack nervous tissue.

While it is quite obvious that acetone, beta-oxybutyric acid or aceto-acetic acid exercise no decided selective action on the papillomacular bundle of the optic nerve, yet it appears reasonable that in certain cases it may produce a retrobulbar neuritis, and that these factors should be taken into consideration in the search for possible etiologic factors in cases of retrobulbar neuritis of otherwise obscure origin.

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[1] Von Noorden: New Aspects of Diabetes, E. B. Treat & Co., New York, 1913, p. 127.

575 Delaware Avenue.

# MEMORANDA

# CHRONIC OCULAR TUBERCULOSIS

## AUTOPSY FINDINGS IN A CASE IN WHICH DEATH WAS DUE TO TUBERCULOSIS OF THE HYPOPHYSIS CEREBRI *

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BOSTON

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Ocular tuberculosis may be classified into at least two well-defined types, the acute and chronic. To the acute type belong the cases of solitary tubercle of the eye, diffuse tuberculosis of the chorioid, acute tuberculous iritis, etc. These cases run a relatively rapid and malignant course, loss of the eye and often also death of the patient ensuing. Although this type of tuberculosis is rare, the eyes, for obvious reasons, have frequently been examined microscopically. The lesions do not differ from active tuberculous lesions elsewhere in the body, and there is no doubt that they result from direct blood metastases. To this type belongs also miliary tuberculosis of the chorioid, which is usually a terminal manifestation of the systemic disease. Intermediate between cases of the acute and chronic type of ocular tuberculosis are certain cases of tuberculous choroiditis which are also probably due to direct blood metastases.

To the chronic type of ocular tuberculosis belong the far more frequent cases of tuberculous scleritis, keratitis, chronic iritis, and cyclitis. These cases run an extremely slow course, are almost never fatal, and healing ultimately occurs, followed frequently by recurrences. Both eyes are most often involved, though not always simultaneously. In 1907 I showed by means of tuberculin tests and microscopic examinations of excised nodules that tuberculous scleritis, the most common manifestation of chronic ocular tuberculosis, is in reality identical with anterior nodular

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scleritis.[1] This condition had generally been designated simply as scleritis or episcleritis and regarded as rheumatic in nature. More recently, I have brought forward evidence that the lesions of chronic tuberculous scleritis, keratitis, iritis, cyclitis, and sometimes, choroiditis, have the same origin, which, moreover, as will be pointed out, is essentially different from that of acute ocular tuberculosis.[2]

There are still other affections of the eye that may be due directly or indirectly to tuberculosis but concerning which our knowledge is yet highly imperfect. Thus, phlyctenulosis is generally believed to be due to sytemic tuberculosis, but on evidence that is not accepted as conclusive. 'Recurrent hemorrhages of the retina and vitreous, exudative retinitis (Coats), and periphlebitis retinalis adolescentium (Igersheimer) all may prove to be manifestations of tuberculosis. It is noteworthy that these conditions in general occur at an earlier period of life than does chronic ocular tuberculosis.

Microscopic examinations of entire eyes from cases of chronic ocular tuberculosis have been few, while so far as I know a general autopsy in such a case has never been recorded.[3] The case that I have to report is therefore especially noteworthy, since not only were both eyes obtained for examination but a general autopsy was performed. It also seems to be only the third case yet recorded of death due to tuberculosis of the hypophysis cerebri.

## REPORT OF CASE

J. M., married woman, aged 36, multipara, was first admitted to the Massachusetts Charitable Eye and Ear Infirmary Jan. 18, 1912, for a tuberculin test. She was readmitted Feb. 24, 1913, and discharged March 8, 1913. April 4, 1913, she was again admitted, at which time she came under my care until her death.

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[1] Verhoeff, F. H.: Tuberculous Scleritis, a Commonly Unrecognized Form of Tuberculosis, Boston Med. and Surg. Jour., March 14, 1907, p. 317.

[2] Verhoeff, F. H.: The Histologic Findings in a Case of Tuberculous Cyclitis, and a Theory as to the Origin of Tuberculous Scleritis and Keratitis, Tr. Am. Ophth. Soc., 1910, p. 566; The Experimental Production of Sclerokeratitis and Chronic Intra-Ocular Tuberculosis, Arch. Ophth., 1913, xlii, 471.

[3] Hayashi (Ueber einen bemerkenswerten Fall von Tuberkulose des Auges mit Sektions-Befund, Klin. Monatsbl. f. Augenh., 1911, xlix, 274) reported a case that on clinical grounds he regarded as probably tuberculous, in which a general autopsy was performed. Inasmuch, however, as the microscopic examination of the eyes gave no evidences of tuberculosis, and no tuberculous lesions were found elsewhere, this case certainly cannot be accepted as one of tuberculosis.

*Ocular History and Condition.*—The right eye first became affected with keratitis three years before death. This subsided after one year, leaving the vision of the right eye reduced to 20/100; it remained so for one year. The left eye then became similarly affected and soon afterward the right eye again became inflamed, after which neither eye was entirely free from inflammation. Seven weeks before death, visual acuity of the right eye was 3/200, and of the left eye 2/200. At this time the right eye was considerably congested the cornea was vascularized, generally hazy, and showed scars of old sclerokeratitis above, and two deep interstitial opacities in inner and outer lower quadrants respectively, each about 1.75 mm. in diameter. In addition, near the limbus below there was a large nodule which projected into the anterior chamber. The left eye was almost free from congestion but showed a condition of the cornea similar to that of the right eye, with the exception of the nodule projecting into the anterior chamber. There were no nodules of scleritis visible in either eye. Each eye showed a few small posterior synechiae, but the pupils dilated fairly well under atropine. The anterior chambers were of normal depths, and the tension of each was not elevated. Fundus details could not be made out owing to the corneal opacities. Paracentesis of the cornea was done three times on the right eye, and each time was followed by noticeable clearing of the corneal haze and decrease in the congestion, but the effect was not lasting.

*General History and Condition.*—The general condition of the patient was good until the year of her death. Tuberculin tests made four months before death (1, 5, and 10 mg. of old tuberculin) gave no definite focal or general reactions, the highest temperature reached being 99.4 F. This, however, was about 2 degrees above the usual temperature, which was persistently low, averaging about 97 F. and often falling below 96 F. in the morning. A general physical examination made at the Massachusetts General Hospital April 8, 1913, showed double mitral disease of the heart, but was otherwise negative. The anterior cervical lymph-glands were slightly enlarged. Eight months before death the menses permanently ceased. The patient had always been mentally bright and cheerful, but during her final stay in the hospital she became markedly dull and apathetic. During this time it was noted that she had polydipsia and marked polyuria, but her appetite was very poor. Examination of the urine was negative for sugar and albumin. The pulse-rate showed extreme variations, at times in the morning falling to 60 and in the evening rising to 120. Toward the end it became more constant, varying between 70 and 80 until a few days before death.

The respiration rate varied between 15 and 20. The patient was habitually constipated, but about ten days before death she developed diarrhea and pain in the bowels. This confined her to bed for three days, when she improved sufficiently to walk about. The day before her death, she was attacked by still more severe diarrhea and pain, accompanied by attacks of syncope. Finally she became comatose, and died May 29, 1913. Three days before death her temperature began to rise slightly, reaching 100.5 just before death, with a pulse-rate of 110.

*Necropsy.*—By Dr. Verhoeff, eight hours after death.—Body that of a fairly well-nourished woman. Peritoneal and pleural cavities free from fluid. Pericardial cavity does not contain an excess of fluid. Lungs: On the right side the pleural cavity is partly obliterated by old adhesions extending from the base of the lower lobe. Along the anterior margin of the base of the right lower lobe immediately beneath the adherent pleura, there are three contiguous nodules each about 8 mm. in diameter, and the lung margin is here more or less thickened for a distance of 5 cm. This is evidently a tuberculous process. On section, no other evidences of tuberculosis, either old or recent, can be made out after the most careful examination. The apices are emphysematous only. There is marked anthracosis. The bronchial glands are pigmented and slightly enlarged, but are not caseous on section. Heart: Weight, 220 gm. Thickness of wall of left ventricle; 14 mm.; of right, 6 mm. Ventricles not abnor-

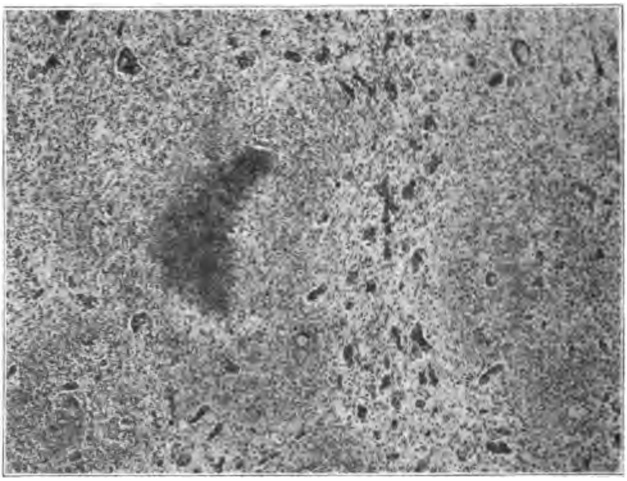

Fig. 1.—Tuberculosis of the hypophysis cerebri, showing giant cells, caseation and remains of parenchyma of anterior lobe; × 40.

mally dilated. The tricuspid and mitral valves are thickened and show a few old vegetations. The aortic valves are also slightly thickened. The pulmonary valves are normal. The foramen ovale is not patent. The aorta and coronary arteries are normal.

The spleen, kidneys, liver, bladder, stomach, uterus, fallopian tubes, and ovaries are normal. The mesenteric glands are in general very slightly enlarged, the largest being 1 cm. in diameter, and those beneath the diaphragm are pigmented. The solitary lymph-nodes and Peyer's patches of the intestines are abnormally large for an adult, the individual nodes measuring from 0.5 mm. to 2 mm. in size, and are unusually numerous. Otherwise the intestines are normal.

Head: The meninges are normal. The hypophysis cerebri is enlarged to about ten times the volume of a normal hypophysis, measuring 21 mm. by 18 mm. by 15 mm. It is firmly united with the chiasm which it has carried backward. The brain on section is normal. The ventricles and chorioid plexus, examined with special care, are free from tubercles.

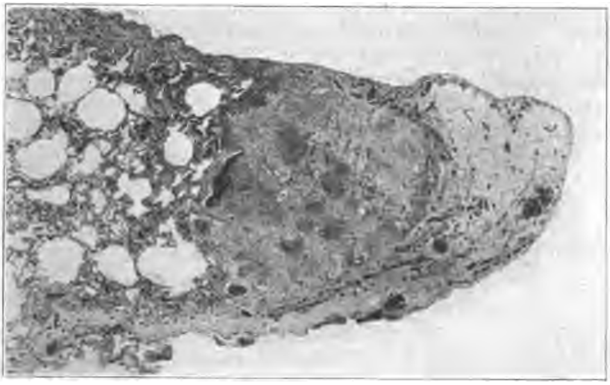

Fig. 2.—Tuberculous focus at base of right lung; × 6.

*Histologic Examination.*—The liver, spleen, and ovaries are normal. The kidneys show slight subacute glomerulonephritis (?). The intestines show moderate hyperplasia of the lymph follicles. The bronchial lymph-glands show the usual reaction to carbon pigment, but are not tuberculous. The mesenteric lymph-glands also show no evidences of tuberculosis.

Lung: The process at the base of the right lung shows the typical picture of tuberculosis, with foci of epithelioid and giant cells, a considerable amount of fibrin, and small areas of caseation (Fig. 2). There is notably slight lymphoid cell infiltration, and the areas of caseation are not infiltrated with pus cells. Some of the individual tubercles consist of discrete endothelial cells situated within distended alveoli. Most of them, however, show a syncytial structure and are undergoing fibrosis. The conglomerate tubercle as a whole contains a large amount of fibrous tissue. About 16 mm. from the main process, and 3 mm. beneath the pleura, there are three-minute tubercles that appear to be of relatively recent origin. The pleura over the tuberculous process and in the vicinity of the latter is not itself tuberculous, but consists of a thick layer of fibrous tissue containing in places large masses of adipose tissue. The lung tissue beneath the thickened pleura shows, in addition to the tuberculous process, the usual changes associated with anthracosis, and is emphysematous.

The hypophysis cerebri has been converted into a large tubercle of the fibroid type with a central caseous center about 5 mm. in diameter. Not a single area of non-tuberculous hypophyseal tissue can be found anywhere, but remains of the acini of the anterior lobe are scattered here and there (Fig. 1). Small discrete tubercles are numerous. These always consist of a syncytium of endothelial cells often containing Langhans' giant cells, and many of them show caseation. The large central area of caseation and the smaller areas as well are free from purulent infiltration but contain an abundance of reticulated fibrin. Lymphoid cell infiltration in general is slight, but in places is fairly marked. From the hypophysis the tuberculous process has slightly invaded the chiasm. The latter also shows interstitial infiltration with lymphoid cells and an occasional deeply seated tubercle. The infundibulum is normal except at its junction with the hypophysis, where it shows an inflammatory reaction.

## PATHOLOGIC EXAMINATION OF THE EYES

Both eyes were removed at autopsy and opened by equatorial section. The right eye was fixed in 10 per cent. liquor formaldehydi, but before it was placed in this solution a piece of the ciliary body, as described below, was removed for animal inoculation. The left eye was fixed in Zenker's fluid.

*Right Eye.*—The globe is normal in size and shape. The cornea shows the scars already noted clinically. The nodule projecting into the anterior chamber from the cornea is ovoid in shape, situated parallel to, and 1.5 mm. from the canal of Schlemm, and measures 1 mm. by 1.5 mm. by 4 mm. in size. The anterior chamber is free from coagulum. The lens is *in situ* and clear. There is complete peripheral anterior synechia. The pupil, 4 mm. in diameter, shows six delicate posterior synechiae. The vitreous humor is of normal consistence, and clear in its posterior portion. In its anterior portion it shows a number of dots scattered within it. The largest of these, about 1 mm. in diameter, are twelve in number. The ciliary body shows four white spots irregular in shape, situated just behind the ciliary processes. Three of these are below, 2 mm. apart, the largest being 2 mm. in diameter. The other 0.75 mm. in diameter, is on the nasal side. The chorioid and retina are *in situ* and normal. The optic disk is not cupped.

*Histologic Examination of Right Eye.*—For a distance of about 3 mm. from the limbus above, the cornea has been converted into vascularized scar tissue, but it here contains no definite tuberculous foci. The central portion of the cornea is relatively normal, but shows occasional vessels in its posterior layers. Near the center, an old non-vascular scar is found just beneath Bowman's membrane. The epithelium is everywhere intact. Bowman's membrane is also intact except over the scar near the limbus above, where it has been destroyed in places. Descemet's membrane and the endothelium are normal except at the site of the nodule below. This nodule (Fig. 3) is found to be a large tubercle that has involved the posterior layers of the cornea only to a depth of five or six lamellae. Some sections show curled fragments of Descemet's membrane carried into the cornea by the tubercle. The structure of the tubercle is unusual. Within the cornea and at its base it is composed of epithelioid and giant cells as is usual, but its greatest mass, lying within the anterior chamber, is composed largely of fibrin. The fibrin is covered with a thick layer of fibroblasts, and the latter are also invading it from the base. It is thus evident that the large size and unusual structure of the tubercle is due to the deposition of fibrin on a tuberculous focus primarily involving the inner surface of the cornea. Whether this focus originated inside or outside of Descemet's membrane it is now impossible to determine.

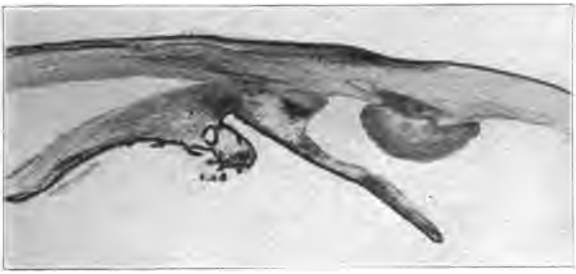

Fig. 3.—Tuberculous scleritis; tubercle in filtration angle, and large tubercle of unusual character on posterior surface of cornea; × 10.

Filtration Angle: This is blocked by the root of the iris all around, and almost every section shows it to contain one or more small tuberculous foci, most of which are in various stages of fibrosis (Fig. 3). Sometimes a focus is seen extending into the cornea beneath Descemet's membrane.

Sclera: In the vicinity of the limbus above, the sclera is abnormally vascularized and largely replaced by fibrous tissue continuous with that of the cornea (old sclerokeratitis). In addition, it shows marked perivascular infiltration with lymphoid cells and a number of small stretched out areas of epithelioid cells, usually in the middle layers. Below, the sclera is less cicatrized, and here also shows tuberculous foci (Fig. 3). These are more active or recent than those above, and one of them, composed largely of discrete endothelial cells, is almost in contact with the conjunctival epithelium.

Iris: The iris shows diffuse infiltration with chronic inflammatory cells, chiefly lymphocytes, and also numerous sharply defined lymphocytic nodules. The latter contain no germinal centers as do true lymph follicles. Attached to the pupillary margin in places there are minute wedge-shaped foci of epithelial cells—tuberculous posterior synechiae (Fig. 4).

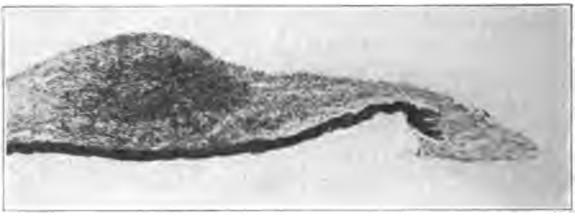

Fig. 4.—Tuberculous synechia at pupillary margin of iris; lymphoid nodule in iris stroma; × 30.

Ciliary Body: In general, the ciliary body is almost normal. It shows only slight lymphocytic infiltration, but is giving off a considerable number of lymphocytes from its surface. In places there is slight proliferation of the pars plana ciliaris retinae. The white spots on the pars plana are found to be localized tuberculous foci involving chiefly the surface (Figs. 5 and 6). They consist of delicate avascular hyaline tissue in which more or less circumscribed areas of epithelioid cells occur. Each is intimately connected with the epithelium, which is greatly altered beneath it. In some places the epithelium is almost completely destroyed by foci of epithelioid cells which have penetrated it and slightly involved the underlying stroma. In other places its two layers are split apart. In the upper part of the eye a small tuberculous focus is found beneath the intact epithelium over which there is no tuberculous process. This focus also differs from the others in being surrounded by a marked lymphocytic infiltration.

Vitreous Humor: The white dots are found to be tubercles, free from caseation, composed of epithelioid cells (Fig. 8).

Chorioid: This is normal.

Retina: So far as can be determined the retina is normal. The ganglion cells are present in normal numbers but show post-mortem changes.

Optic Nerve: The optic disk is not definitely cupped, but the lamina cribrosa is curved backward, and there are small cavernous spaces just in front of it. The myelin sheath stain shows no atrophy of the nerve stem.

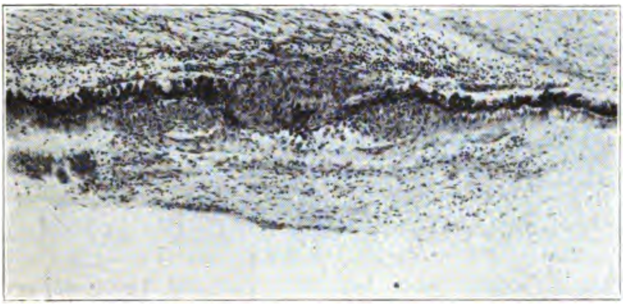

Fig. 5.—Tuberculous focus involving surface of ciliary body. The epithelium has been broken through. Macroscopically this appeared as a white spot about 1.5 mm. in diameter; × 50.

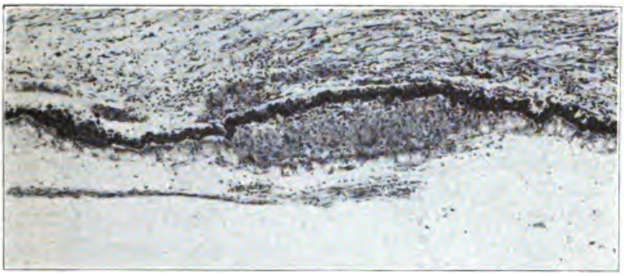

Fig. 6.—Tuberculous focus involving only the epithelium of the ciliary body. A giant cell is present; × 50.

*Left Eye.*—This is also normal in size and shape. The anterior chamber is free from coagulum. There is complete peripheral anterior synechia. The lens is *in situ* and clear. The ciliary body shows one minute white spot on the pars plana ciliaris, but is otherwise apparently normal. The vitreous humor is free from tubercles and otherwise normal. The retina and chorioid are *in situ*, and normal. The optic disk is not cupped.

*Histologic Examination of Left Eye.*—The left eye shows changes that differ only in degree from those in the right. The older lesions, represented by scars in the cornea and sclera, are less well marked. Some of the old foci in the filtration angle, however, have become almost completely converted into hyaline tissue and thus show further retrogression than any corresponding foci in the right eye. The active tuberculous foci, both within the filtration angle and elsewhere, are more recent than those in the right eye, as indicated by the fact that the cells are more discrete. Numerous active foci are found in the anterior sclera both above and below, and a small one especially active is found just beneath the epithelium at the limbus. One active focus is seen in the cornea situated immediately beneath Descemet's membrane. Active foci are also found within and beneath the hyaline tissue in the filtration angle from which they sometimes extend into the cornea. The iris shows numerous tuberculous synechiae, and in addition small tubercles within the

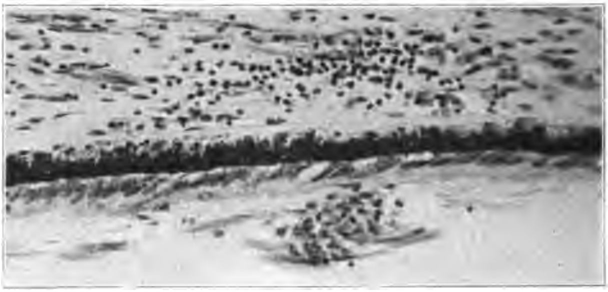

Fig. 7.—Minute tubercle composed of endothelial cells, on surface of ciliary body; × 195.

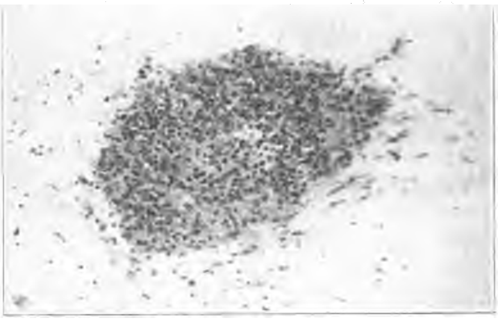

Fig. 8.—Small tubercle in anterior part of vitreous humor; × 145.

stroma near the pupillary margin. The iris is free from the lymphocytic nodules seen in the right eye. In fact, the eye as a whole shows much less lymphocytic infiltration than the right eye.

The ciliary body shows a few small recent foci on its surface, but none of such size or so old as those in the right eye. Below there is a minute tubercle 0.1 mm. in size, composed of discrete endothelial cells adherent to the pars plana (Fig. 7). In another place there is a disk-shaped nodule 0.4 mm. in diameter on the surface of the pars plana, while beneath the epithelium there are two small tubercles. In still another place there is a small tubercle in the stroma that is just beginning to invade the epithelium.

The retina, chorioid, and optic nerve are free from tuberculous foci and present the same appearance as in the right eye.

### BACTERIOLOGIC EXAMINATION

Numerous sections stained for tubercle bacilli show no bacilli in the lesions of the eyes, hypophysis,, or lungs. Spreads from the tubercles of the vitreous humor, made before fixation, are also negative for bacilli. A piece of the ciliary body from the right eye containing the largest one of the tuberculous foci, was introduced into the anterior chamber of a rabbit's eye. This adhered to the surface of the iris but was completely absorbed without producing tuberculosis.

*Pathologic Diagnosis.*—Tuberculosis of the hypophysis cerebri. Old pleural adhesions. Small conglomerate tubercle, base of right lung. Old endocarditis. Hyperplasia of lymph follicles of intestines. Chronic bilateral tuberculous scleritis, keratitis, iritis and cyclitis. Tubercles of the vitreous humor.

In spite of the inconclusive tuberculin test and the negative bacteriologic findings, there can be no doubt that this was a case of tuberculosis. The lesions in the lung, hypophysis, and eyes were typical, mutually corroborative, and incapable of any other interpretation. Moreover, the ocular history and clinical picture were perfectly characteristic of chronic ocular tuberculosis.

This case proves, so far as it is possible for a single case to do so, that complete destruction of the hypophysis cerebri is incompatible with life, for there can be no doubt that death was due to loss of function of the hypophysis resulting from its complete tuberculous involvement, since at autopsy no other cause for death could be found. In other words, this was an extreme case of hypopituitarism. Unfortunately, this fact was not recognized during the life of the patient, and the case was not treated or investigated from this point of view. Some of the more important clinical symptoms, however, were noted. These were the cessation of the menses eight months before death, polyuria and polydipsia, and marked mental dulness. There was not undue adiposity, though the amount of subcutaneous fat was greater than would have been expected in view of the little nourishment the patient could be induced to take. The persistent low temperature was characteristic of hypopituitarism, but was equally so of chronic ocular tuberculosis. The terminal symptoms of hypopituitarism have not been described in a series of cases, so that whether or not those observed in this case can be regarded as characteristic is uncertain. The attacks of syncope and the gradual onset of coma might have been expected. The diarrhea and abdominal pain, both probably due to exaggerated peristalsis, beginning ten days before death, were perhaps analogous to the hiccoughs of Addison's disease.

## CASES IN LITERATURE

In the literature I have been able to find reports of only eight cases of hypophyseal tuberculosis, none of which was associated with ocular involvement.

Hueter[4]and Heidkamp[5] describe one case each of generalized tuberculosis with death from tuberculosis meningitis, associated with tuberculosis of the hypophysis. In Sommer's[6] case—this was also one in which death occurred from tuberculous meningitis—the hypophyseal involvement was probably by extension from tuberculosis of the sphenoid bone. In the case of Lancereaux[7], death was due to pulmonary and intestinal tuberculosis, and the hypophysis was found enlarged to double normal size and pervaded by miliary granulations. The case of Lucien and Parisot[8] was one of diabetes mellitus with death from subarachnoid hemorrhage, in which old pulmonary tuberculosis and miliary tuberculosis of the hypophysis were found. In Beck's[9] case, in which death was due to sepsis, the hypophysis had been converted into a large

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4. Hueter, C.: Hypophysistuberkulose bei einer Zwergin, Virchows Arch. f. path. Anat., 1905, clxxxii, 219.

5. Heidkamp, Hans: Beitrag zur Tuberkulose der Hypophyse, Virchows Arch. f. path. Anat., 1912, ccx, 445.

6. Sommer, H.: Zur Tuberkulose der Hypophyse, Ztschr. f. Laryngol., 1910, ii, 355.

7. Lancereaux, E.: Traité d'anatomie pathologique, 1883; Cited by Heidkamp, Note 5.

8. Lucien, M., and Parisot, J.: Tuberkulose de l'hypophyse et diabète sucré, Rev. neurol., 1909, No. 16, p. 970.

9. Beck: Tuberkulose Entartung des Hirnanhanges als Ursache der Diplopie und des Strabismus, Ztschr. f. d. Ophth., 1835, iv, 401.

encapsulated tubercle, and there were no other evidences of tuberculosis.

Wagner's[10] case was that of a scrofulous girl aged 13, with ozena, slight glandular swelling, and attacks of conjunctivitis. There was sudden onset of convulsions followed by coma and death within six hours. At autopsy the hypophysis was found completely converted into a tubercle. There were no other tuberculous lesions, but the right lung showed extensive pleural adhesions. (Hueter regarded this case as probably one of gumma of the hypophysis.)

In the case of Haushalter and Lucien,[11] the patient was a girl aged 6½ years. There was marked polyuria and polydipsia, but the urine was free from albumin and sugar. The child was in good spirits. The temperature was never above normal; the pulse-rate was 100. The skin was rough and dry. There was sudden onset of convulsions which lasted fifteen minutes, followed by coma and death. At autopsy the tuber cinereum was found replaced by a large tubercle which had extensively involved the hypophysis by way of the infundibulum. No tubercle bacilli could be found. There was a caseous gland the size of an almond to the right of the trachea and a fibrous tubercle at the apex of the right lung. The latter also showed adherent pleura posteriorly.

In only the last two of these cases could death be attributed to the involvement of the hypophysis. In both, as in my case, death occurred suddenly and unexpectedly. In both there were convulsions, however, whereas in my case these were absent. This may have been due to the fact that these cases were in children in whom convulsions readily occur, or to the fact that the destruction of the hypophysis was more rapid than in my case. Only in the case of Haushalter and Lucien were diabetes insipidus and polydipsia noted, but it is possible that they were present in some of the other cases and overlooked.

The question of the relation of simple polyuria to lesions of the hypophysis is a complicated one. Schafer and Magnus have shown that the injection of extracts of the posterior lobe into animals produces temporary polyuria. Cushing's[12] researches also indicate that polyuria of hypophyseal origin is due to involvement of the posterior lobe, but his experiments are appar-

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[10] Wagner, E.: Tuberkel der Glandula pituitaria, Arch. d. Heilk., 1862, iii, 381.

[11] Haushalter, P., and Lucien, M.: Polyurie simple et tubercle de l'hypophyse, Rev. neurol., 1908, p. 1.

[12] Cushing, Harvey: Concerning Diabetes Insipidus and the Polyurias of Hypophyseal Origin, Bull. Mass. Med. Soc., 1913, xxlv, 25.

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ently contradictory as to whether it results from overactivity or underactivity of the latter. Thus he finds that manipulation of this lobe produces temporary, whereas its complete removal causes prolonged polyuria. The present case, as well as that of Haushalter and Lucien, would seem to prove that depressed activity of the hypophysis may cause polyuria. This is supported by that fact that clinical observations show that simple polyuria is associated with symptoms of hypopituitarism rather than with those of hyperpituitarism. Possibly the relatively transient polyuria that results from a sudden excess of pituitary secretion in the blood may be produced in an entirely different way from that of the persistent polyuria of hypopituitarism.

An interesting question concerns the possible relationship, in the present case, of the hypophyseal, pulmonary, and ocular lesions. Judging by the time of onset of clinical symptoms, the duration of the ocular tuberculosis was definitely known to be three years. That the process in the hypophysis could have been of equal duration is possible but improbable. Its duration, however, was certainly over eight months, since the first hypophyseal symptom noted, cessation of the menses, occurred eight months before death. That the hypophyseal lesion could have given rise to the ocular tuberculosis by way of the optic nerve is hardly conceivable. The chiasm was only slightly involved at death and there were no foci in the optic nerve or in the chorioid, so that for the eyes to have been infected in this way it would have been necessary for retrograde metastases to have traveled along the subvaginal space and thence along the subchorioidal space into the anterior chamber without producing any lesions in their transit. On the other hand, for similar reasons, it is almost equally improbable that in the reverse way the ocular lesions could have infected the hypophysis. In all probability, therefore, the ocular and hypophyseal lesions were independent of each other, a conclusion that is strengthened by the fact that chronic ocular tuberculosis is relatively common while tuberculosis of the hypophysis is extremely rare.

There can be little doubt that the tuberculous focus which gave rise to the ocular and hypophyseal lesions was the small conglomerate tubercle at the base of the right lung. The latter may have represented the recurrence of an old lesion, perhaps originally associated with the old pleurisy in this situation, for a fresh infection would have been expected to produce a more active type of tuberculosis. The persistent low temperature and the absence of a well-marked tuberculin reaction are likewise against a recent infection, as is also the fact that tubercle bacilli could not be found in sections of any of the lesions. A low order of virulence of the infecting organisms was indicated by the negative result of animal inoculation, although it is possible that the amount of material used for this purpose was insufficient.

As is well known, patients having chronic ocular tuberculosis seldom if ever die from pulmonary or generalized tuberculosis. The present case is not a real exception to this rule, since it is evident that death would not have ensued had it not been for the chance involvement of such an important structure as the hypophysis. Occurring elsewhere, a lesion of such small size in a case of this type would undoubtedly have healed. This case, in fact, affords the first demonstration based on anatomic findings of how insignificant the systemic tuberculosis may be in cases of chronic ocular tuberculosis.

Each eye in this case showed the typical clinical picture of recurrent tuberculous sclerokeratitis. While the case was under my observation there was active keratitis, but the only clinical evidence of scleritis was the presence of the corneal scars extending out from the limbus. Nevertheless, on microscopic examination the anterior part of the sclera in each eye showed numerous active tuberculous foci, a fact that suggests that scleritis may often exist without characteristic clinical manifestations, especially when in association with tuberculous keratitis.

The lesions of the cornea, iris, filtration angle, and surface of the ciliary body were similar to those previously described by me. The tuberculous synechiae at the pupillary margins were especially well marked. The chorioid and retina were free from tubercles. None of the ocular lesions showed caseation, but simply epithelioid and giant cells and more or less fibrosis. The right eye, in addition, showed two features not hitherto described, namely, the unusual type of tubercle on the posterior surface of the cornea, and the tubercles of the vitreous.

Elsewhere I have brought forward evidence, which I believe to be sufficiently conclusive, that in chronic ocular tuberculosis the infection of the ocular tissues takes place from the aqueous humor. The bacilli reach the latter from the blood through the ciliary processes, and are then carried to the filtration angle, whence they may pass into the cornea or sclera. They may also be deposited on the ciliary body or carried into the iris, producing focal lesions here, or into the subchorioidal space, where they may give rise to chorioidal foci. In experimental ocular tuberculosis produced by injecting dead tubercle bacilli into the vitreous or aqueous humors, all these lesions were produced, and in addition sometimes tubercles of the retina and vitreous. It is evident that the ocular lesions in the present case had a similar origin. The vitreous tubercles of the right eye were identical in appearance with those produced experimentally. The foci involving the surfaces of the ciliary body in each eye were clearly due to infection from the aqueous humor, since all stages in their formation could be observed, beginning with a minute nodule of endothelial cells on the surface of the intact epithelium. A few tubercles, however, were found entirely beneath the epithelium; and these might have been regarded as direct blood metastases, were it not for the fact that the experiments referred to demonstrated that minute tubercles can pass through the pars ciliaris retinae into the underlying stroma without leaving permanent defects in the epithelium.

It will be noted that this theory necessitates the assumption, as yet unproved, that tubercle bacilli may pass from the vessels of the ciliary body into the aqueous humor. The evidence for the theory, however, is otherwise so strong, it seems to me, that it may be regarded as sufficient to establish this assumption as a fact. It is known that bacteria may be excreted by the kidneys, so that *a priori* there is no reason why they cannot be excreted by the ciliary processes. If this is so, it may be asked why tubercle bacilli are not similarly excreted in cases of active tuberculosis. This is probably due to the fact that here the conditions of immunity are different from those existing in chronic ocular tuberculosis, so that when bacilli get into the blood they are either quickly destroyed or immediately produce lesions wherever they happen to lodge, which in case of the eye is most often within the chorioid. It may also be asked why other organisms are not sometimes excreted into the aqueous humor. The answer to this is that there is no evidence that under certain conditions of immunity this may not happen, or, in fact, that other forms of iritis and uveitis may not be produced in this way.

A survey of the features of chronic ocular tuberculosis in general, including those of the present case, makes it plain that there is something special about this type of tuberculosis in addition to the mode of origin of the eye lesions. This is indicated among other facts by the following:

1. Chronic ocular tuberculosis almost never occurs in cases in which there are well-marked clinical signs of systemic tuberculosis and in which, therefore, metastases would presumably be most apt to occur. On the contrary, it occurs chiefly in cases in which it is difficult or even impossible to find any other indications of tuberculosis. The present case is in this connection a striking example.

2. Although chronic ocular tuberculosis is rare compared to the incidence of tuberculosis in general, yet when it occurs it is almost always sooner or later bilateral, thus showing plainly that it does not represent chance metastases.

3. It occurs almost exclusively in adults, and sometimes even in patients of advanced age.

4. It occurs far more frequently in females than in males.

5. The temperature is almost never elevated and is often subnormal.

6. A large dose of tuberculin is usually required to produce a constitutional reaction, and rarely a tuberculin reaction cannot be obtained at all.

7. Acid-fast tubercle bacilli seldom if ever can be demonstrated in the lesions.[13]

8. Animal inoculations of the lesions usually if not always give negative results.

To account for the foregoing facts, I venture to advance the following hypothesis. It is, briefly, that a condition is established in which the tissues of the host and the infecting organisms have become relatively tolerant with respect to each other. This condition is due to the fact that the tissues and organisms have existed so long under the same environment that they have developed similar immunities. The mutual tolerance is, however, not absolute, so that lesions of slight intensity but of long duration are produced, and the growth of the organisms is to a greater or less degree inhibited.[14]

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[13] I have found that altered pigment granules may occasionally stain by the carbolfuchsin method and appear not unlike tubercle bacilli. This no doubt has given rise to error.

According to this hypothesis the history of a typical case of chronic ocular tuberculosis may be reconstructed thus: In early life, most often in childhood, the patient becomes infected with tubercle bacilli, perhaps, usually of the bovine type. A sufficient degree of immunity is quickly developed and the lesions heal without having become extensive. Many of the bacilli, however, remain alive in the lesions. In the course of years, the bacilli become more or less active from time to time, owing to varying resistance of the patient, and as a result the tissues and bacilli become more and more tolerant of each other. The bacilli become not only relatively but also absolutely less virulent, and lose their acid-fast property.[15] At some time under these conditions the resistance of the patient becomes depressed to an unusual degree, and the original lesions take on renewed activity. Bacilli are now at times given off into the blood, but owing to their small numbers, lack of virulence and the resistance of the patient, metastatic lesions are directly produced only exceptionally.[16] From time to time, however, bacilli pass from the capillaries of the ciliary body into the aqueous humor, where, owing to the lack of immune substances encountered, they proliferate and then sometimes give rise to the various lesions of chronic ocular tuberculosis, as already described, of which those of scleritis and keratitis are the most frequent. Many times, no doubt, the bacilli pass into the canal of Schlemm and are carried from the eye without producing any lesions. On account of the relative inactivity of the bacilli and diminished sensitiveness of the tissues to tuberculo-toxins, the ocular lesions do not reach large size and are often associated with little or no leukocytic infiltration. The resistance of the patient now becomes reestablished and the lesions heal. After a shorter or longer interval, the resistance again becomes lowered and the preceding process is repeated, with the addition that some of the older ocular lesions again become active.

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[14] This theory, it seems to me, also explains well the parasyphilitic affections. Thus it explains why they are not amenable to mercurial treatment, since the spirochetes and body cells have become to such an extent alike in their vital processes that drugs no longer act differentially on the organisms. Possibly it explains also why hereditary interstitial keratitis is likewise resistant to treatment.

[15] I have shown experimentally that dead tubercle bacilli may lose their acid-fast property when injected into the eyes of immunized animals, and yet remain capable of producing tubercles.

[16] The thought naturally suggests itself here that it is to such relatively benign metastatic lesions that are due certain forms of tuberculous chorioiditis, retinitis, etc.

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# MEMORANDA

# EPITHELIOMA OF THE LIDS *

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Epitheliomas of the lids require some consideration apart from epitheliomas of the skin elsewhere, because of their proximity to the orbit and sinuses and because of the disastrous effects of destruction of the lids on vision and on the appearance of the patient. A good proportion of these patients do not consult the ophthalmologist but are sent to the general surgeon after a long course of salves, pastes and Roentgen-ray treatment.

I have been able to study eighty-eight cases of epithelioma primary in the lids and canthi, or involving them from the skin in the immediate vicinity (nose, cheek, brow), having chiefly in mind the prognosis of operative treatment. The statistical data were collected to serve as a basis of comparison with other methods of treatment, especially radium. It should be added that the diagnosis in all cases was confirmed by microscopic examination.

The epitheliomas of the lid all belonged to the type known as basal-celled, or, to use a rather popular term, rodent ulcer. By this is commonly meant clinically a cancer of the skin of very slow growth, with no tendency to glandular metastases, spreading by continuity, and usually ulcerating as it progresses. Pathologically, it is somewhat more difficult to define, though this is perhaps of no great consequence to our present object. Adami[1] sums up the present view of this class of epithelioma.

> Krompecher, recognizing that all conditions classified as rodent ulcer do not belong to the type, has labeled this form

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

* From the Mayo Clinic.

[1] Adami: General Pathology, Philadelphia and New York, 1908, 735.

basal-celled carcinoma, on the mistaken ground that, as it shows no prickle cells nor keratinization, it is derived wholly from the basal, undifferentiated cells of the rate Malpighii. But this is so also for all epitheliomas. In the more highly developed forms the prickle cells present do not arise from pre-existing prickle cells, but also from basal mother cells. It is the stage of undifferentiation and anaplasia—the capacity to develop beyond a certain point—that determines the form of the cells.

After the growth has progressed, both basal and squamous cells are to be found in the field, one or the other predominating. Sometimes the appearance may closely simulate sarcoma from the tendency of the epithelial cells to assume spindle forms. I have seen this mistaken diagnosis made several times in epitheliomas of the orbit (Figs. 1 and 2).

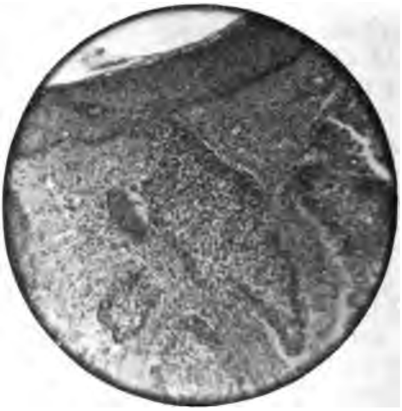

Fig. 1.—Epithelial down growth showing spindle and oval cells which, when in masses, often look like sarcoma cells.

In this series the average age of incidence was 53 years. The youngest patient was 24 years old. The average patient waited five years before coming for treatment; many of them from fifteen to twenty-five years; these latter mostly began as keratoses and small wart-like nodules. Some developed rapidly, that is, in one month.

The favorite points of origin were the lower lid (46 per cent.), the inner canthus, over the lacrimal sac (36 per cent.). The next most common point of origin was the upper lid and, least common, the outer canthus.

Of the cancers of the lid, considerably over half originated in the skin of the lid just away from the cilia, the others starting indifferently on the very margin of the lid or the base. There seems to be no obvious reason why the lids should be so favorable a site for the development of epithelioma. The fact that the lower lid and inner canthus are so much more commonly affected might be attributed to the irritation caused by tears and foreign matter. Certainly these regions are usually hyperemic when the upper lid and outer canthus show comparatively little evidence of irritation. In only one case was the pressure from eye-glasses a probable source of the initial irritation, which is so commonly looked for. The type of skin well recognized as a fertile soil for epithelioma—a dry, rubicund skin, seen most often in blond people exposed to the elements—was very often seen, but by no means predominated.

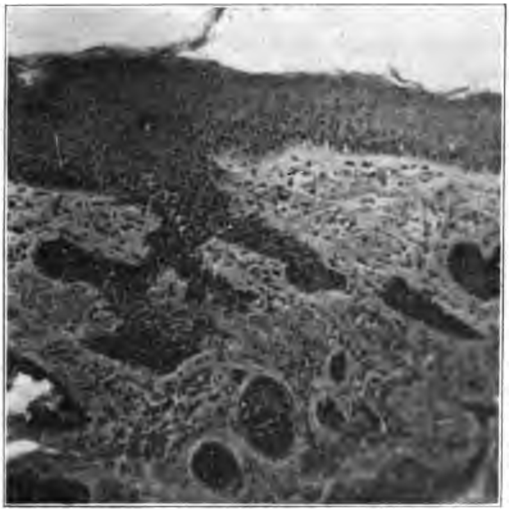

Fig. 2.—The same as Figure 1, except growth is taken from nose.

The majority of these cancers progressed by invasion only, the first step being attachment to the periosteum of the orbital border, then progression into the orbit and conjunctiva. In several cases the inner orbital walls were destroyed. Occasionally the antrum was involved, and more often the ethmoids, rendering successful operation very difficult.

In five cases, cervical or parotid glandular involvement was present when the patient was first seen. These two groups of glands drain all of the skin and the conjunctiva, while the orbit drains into the internal maxillary group and thence into the deep cervical. Unsuccessful treatment seems to favor glandular involvement, since, of these five patients, only one had escaped pastes or Roentgen rays previous to examination in our clinic, and about the same proportion was found in epitheliomas of the cheek with glandular involvement. The type of tumor did not differ microscopically in these cases from that in cases not showing glandular involvement, so that the failure to involve glands is not a necessary characteristic of rodent ulcer. Our pathologic records, however, do not indicate the character of the metastatic growth. None of the patients developed enlarged glands after coming under our observation, which may have been due to the methods of treatment employed.

These patients were treated chiefly in three ways — radical excision, excision with actual cautery of the wound, or simply actual cautery, the method employed depending on the site of the growth and the degree to which it had progressed. Purely cutaneous growths, if small, were cauterized. Growths involving the border of the lid, but not adherent to the periosteum, were excised and a plastic operation done to restore the lid. Where the growth was adherent to periosteum or had involved conjunctiva and orbit, or tear-sac, actual cautery was added to excision. In cases of orbital involvement not readily accessible, an exenteration of the orbit, including the periosteum, was performed and the contents of the apex cauterized. For cauterizing large areas, the soldering iron was found preferable on account of the deep cooking given the tissues — a principle used in Percy's treatment of cancers. Involved sinuses were opened, curetted and cauterized in so far as possible.

The plastic repair of the lower lid is a frequent problem. Many satisfactory results were obtained from the transplantation of a "hammock" flap of skin from the upper lid, a method devised by C. H. Mayo.

This flap is soft, of the same color as the normal lid, and grows readily. The operation recently described by Gibbon[2] promises to be the one of choice when the lower lid has been largely destroyed. No conventional method will serve every case. The repair is easy when a fair amount of the lid has been left. When, however, the entire lid and part of the cheek have been removed, as is often necessary, large flaps from the temple (as noted in the text-books) give the best results. The Dieffenbach and similar procedures, beautiful on paper, have not proved so successful on patients.

## RECURRENCES

This has no reference to final cures, but refers to any recurrence whatever taking place after operation in our clinic. Most of these were small nodules, easily burned off. Of the thirty-three epitheliomas involving the lids and canthi alone (adherent or not adherent), 17 per cent. recurred at some time. These recurrences were all at the site of the previous growth, and not in glands. When, however, the growth had penetrated the orbit, the percentage of recurrence rose rapidly — eight out of eleven. Of thirteen cases involving the orbit, primarily and secondarily, three have passed the one-year period without recurrence. Four others have not yet recurred, but have not gone a year since operation. The time-limit of one year is somewhat arbitrary, but is based on the average time before recurrence, namely, nine months. This is a rather high average, being brought up by a few unusual cases which were reported as having recurred very late. The majority of cases which recurred at all did so in three to four months.

The chief interest in these cases for the clinician lies in the ultimate prognosis. I have not counted as cures cases of less than one year's standing since the last operation or treatment. Naturally, the percentage would be considerably higher if cases of over four months' standing were cited and if all patients were willing to return at the first sign of recurrence.

Of the thirty-three epitheliomas of the lids and canthi, not including the orbit, in which the records have been completed by recent news of the patients, 94 per cent. were cured by operation. When the orbit was involved, the percentage was turned about; 80 per cent. were not permanently cured. These include cases in which the sinuses also were involved. The factors which mitigate against cure are: inner canthus involvement, penetration of conjunctiva or lacrimal sac, orbital involvement and sinus involvement. The cases of glandular involvement cannot be classified because our records are not completed, owing to the lack of recent information of these patients. The one "completed" case had no recurrence after a radical local removal, and dissection of the cervical glands.

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[2] Gibbon: Blepharoplasty by a Pregrafted Flap, read before the American Surgical Association, April 9, 1914.

The comparison of these results with those obtained by the use of radium or Roentgen rays or pastes is difficult because of the absence in most papers on the subject of definite data regarding the duration of the cure. Williams and Ellsworth, however, are not remiss in this regard.[3] Of the nineteen cancers of the skin of the lids, two-year cures are reported in thirteen cases, or 68 per cent. These are evidently not intended to include cases with orbital involvement, since one case of orbital involvement is listed separately. The percentage of two-year cures by operation in our series was 84.

Pinch,[4] in a report of the Radium Institute of London, classified his cases of rodent ulcer into apparently cured, thirty-one; "improved," forty-one; not improved, twelve; abandoned treatment, six. Counting improved, not improved and abandoned as failures (as was done in our series), the percentage of cure is far from satisfactory (about 39 per cent.) and this without any minimum time-limit set. It should be said, however, that these were rodent ulcers of various regions and severities, so that the comparison is not altogether fair. Pinch was able to state on this showing that "rodent ulcer is of all cancers the most amenable to radium treatment." Turner[5] cured six out of nine completed cases of rodent ulcer of various parts of face and lips, or 66 per cent., time not stated. The best opinion at present concerning radium versus operative treatment of these cases is well-summed up by Knox.[6]

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[3] Williams and Ellsworth: Treatment of Superficial New Growth by Pure Radium Bromid, The Journal A. M. A., May 31, 1913, p. 1694.

[4] Pinch: Brit. Med. Jour., Jan. 25, 1913, p. 149.

[5] Turner: Brit. Med. Jour., March 22, 1913, p. 606.

> "1. In all cases of early cancer, the operative method is undoubtedly best; it is quicker, safer and offers the best prospect of cure. . . . 3. In patients who refuse operation or are otherwise unfit for operation, radium is a useful remedy."

Comparison of operative treatment with pastes is also difficult. I have found no recent records sufficiently exact to afford a statistical comparison. Perhaps the most revolting, because easily avoidable, pictures of malpractice that one sees are cases of cancer of the skin treated with various pastes, and the inevitable impression received is that the public has suffered unspeakably from the exploitation of the paste treatment. Nevertheless, it must be acknowledged that in the hands of persons practiced in this procedure, many surprising cures are obtained, and these sometimes in apparently inoperable cases. No doubt the continued financial success of certain "cancer institutes" is maintained by a fair number at least of noteworthy cures.

The Roentgen ray is believed to owe what efficiency it possesses to a ray corresponding to the gamma ray of radium, so that there seems to be no reason for preferring it to the latter (save its greater accessibility) since the curative rays are acknowledged to comprise only a small part of the whole Roentgen ray. In point of fact, the Roentgen ray cannot be regarded as a dependable agent in the treatment of epitheliomas, however useful it may be as a palliative or postoperative measure.

The present evidence seems conclusive that operative measures are, in general, the safest means of treatment we possess for epitheliomas of the lids (and for that matter in any other region), and especially when the growth is rapid and adherence to periosteum or involvement of the orbit has taken place or evidently will soon take place. On the other hand, when the patient can be kept under observation until a cure is completed and the growth is proceeding slowly, there seems to be no good reason why radium should not be used. In cases of this kind, however, it has the disadvantage of slowness, and there is also the difficulty, in most places, of obtaining the radium. But many

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6. Knox: Brit. Med. Jour., June 7, 1913, p. 1196.

patients prefer any amount of treatment to the use of the knife, a psychological factor that is heavily featured in the advertisements of the clever charlatans. The cosmetic results of successful radium therapy are excellent; I have seen no injury or reports of injury to the eye from its use; it is practically painless. In this as in any therapeutic or surgical measure, the experience and ability of the physician in charge plays an important rôle, especially in pioneer work with comparatively new methods of treatment.

The importance of early and radical operation in epitheliomas with orbital extension is well emphasized in the statistical data given above. Unless the growth is so clearly circumscribed that complete removal without sacrificing the eye is certain, a complete exenteration of the orbit followed by cautery is imperative. Extension to the sinuses renders the prognosis nearly hopeless; in such cases, if operation is decided on, it should be followed by the persistent use of radium or by Roentgen ray if the former is not available. Glandular involvement, without orbital extension, does not necessarily indicate a bad prognosis, granting that a proper resection of the gland-bearing fascia be made.

# MEMORANDA

# MEMORANDA

# HEREDITARY GLAUCOMA (SIMPLEX)

## THE REPORT, WITH OPERATIVE NOTES, OF THREE GENERATIONS OF ONE FAMILY [*]

F. PHINIZY CALHOUN, A.B., M.D.
ATLANTA, GA.

Heredity is recognized as a common factor in the causation of glaucoma, both of the inflammatory and non-inflammatory types. As a hereditary type glaucoma simplex is exceptionally rare beyond the second generation, and in my exhaustive search I have been able to find recorded only four other cases or families. The earliest cases are mentioned by Benedict in his publication in 1842; von Graefe in 1869 mentioned the importance of heredity in the causation of glaucoma, although no specific cases were given. Much literature is found on the direct inheritance of glaucoma of the acute or inflammatory type and special mention should be given Harlan's family, where members of five generations were afflicted. The discussion of this type of glaucoma is purposely omitted from this monograph. Lawford's very excellent review of the subject in 1907 in his report of "Examples of Hereditary Primary Glaucoma," has been kindly placed in my hands by the author and I have taken the liberty of quoting freely from the text and of copying his pedigrees.

I have had an unusual opportunity to study clinically this type of glaucoma in a family in which I found no peculiarities, common ailments (other than malaria and glaucoma) or serious illness, and in which eight members are blind or going blind from this mysterious disease. "Anticipation," so prominently mentioned by von Graefe and Lawford, is as prominently displayed in the family which I present.

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[*] This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

[*] Much confusion exists in the selection of titles given papers on the subject of glaucoma and no index could properly classify them. It would be most simple to adhere to the old classification, namely, primary glaucoma, including the acute and chronic inflammatory types; glaucoma simplex, the non-inflammatory type, and secondary glaucoma.

## REPORT OF CASES

Case 1.—This interesting family commences with W. F. H., aged 59, seen by request March 3, 1911. He states positively that his father and mother and other near relatives whom he knew had good sight. The patient always enjoyed good health, having worked on a railroad and farm all of his life. When 29 years of age he was struck in the right eye with a twig, and soon afterward discovered that he could not see well with the left eye. He consulted a competent ophthalmologist six months later, who told him that he had glaucoma in each eye. An iridectomy was made in the left eye, and later the right eye was enucleated on account of pain. Since then he has been able to do his work in a moderate way, although his sight gradually failed until now he is almost blind. Examination: Left eye, cystoid cicatrix in region of old sclera incision. Pupil widely dilated; grey atrophic iris, media clear and marked cupping of vessels, with their caliber much reduced; nerve bluish white. Horizontal diameter of the cornea, 12 mm. Tension + 2. Light perception only.

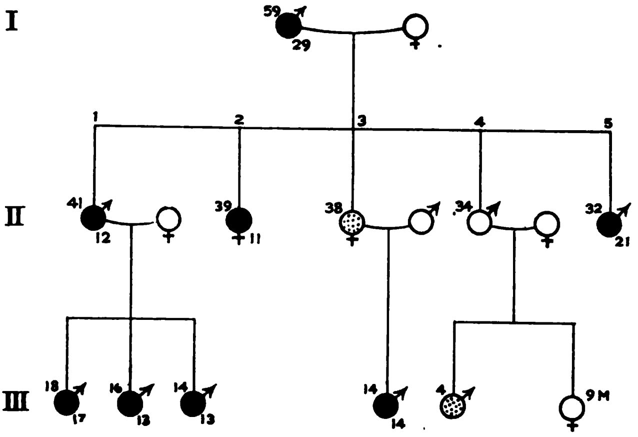

Fig. 1.—Pedigree of author's patient. Explanation of signs employed in this and the following pedigrees: ♂ male; ♀ female; the same characters in black indicate affected persons; the dotted characters persons with amblyopia and doubtful glaucoma. The numbers above each individual indicate the present age; below, the age when bad sight was first detected. Generations are indicated by Roman numerals, I, II, III, etc.

Case 2.—Mrs. S. C. G., aged 41, seen by request July, 1912. Her past history has been good, pneumonia her only serious illness. She first noticed defective sight in the right eye at 12, and later her sight became so bad that she stumbled over chairs and other objects about the house. She never saw halos around lights or had pain in the eyes. At 15 an iridectomy was made on the right eye and soon afterward on the left. She states that the operations have not benefited her. Examination: right eye, vision 0; ciliary staphyloma in the region of old scleral wound; tension +3; horizontal diameter of cornea 11 mm.; iris drawn up in staphyloma; good black pupil and media clear; bluish-white nerve with marked excavation; vessels thread-like; external squint 15°. Left eye, vision 5/200 unimproved; tension +1; corneal diameter 11 mm.; anterior chamber very deep; media clear; atrophic nerve with excavation; vessels small. Color of irises brown. Three months later: Tonometer, right, 11 mm. Hg; left, 16 mm. Hg. Refraction, left, —3 S. She complains that when "bilious" she sees spangles of light; staphyloma more prominent, which accounts for low tension.

Case 3.—D. G., first son of Patient 2, aged 18, was seen Aug. 16, 1909. His health has always been good and six weeks before visit he discovered that while out hunting he could not see well with the right eye; thinks it has been growing worse since. There has been some deep pain in the eye. Examination: Right eye 20/100 unimproved; anterior chamber deep; peculiar physiologic cupping, with a serpentine twist of vessels over the nerve, which at its center dips slightly; field, slight contraction nasally; tension (digital) normal. Left eye, vision 20/20 unimproved, but reads best at normal distance with +1.25 S; eye apparently normal; field normal. Irises brown; diameter of corneas 11.5 mm. The patient passed from notice until Feb. 10, 1911. Vision, right 20/70; vision, left 20/50. Field, marked contraction nasally; tension each eye +1. (For chart of vision and tension see Fig. 2.)

February 21: LaGrange sclerectomy with broad iridectomy right eye. Patient was allowed to go home immediately. Ultimate result satisfactory. March 15: Vision, right, 20/200 — 1 ○ — 1 × 90 = 20/70; tertian malaria.

March 16: Iridectomy (left) section made with thin knife. Patient allowed to leave. Healing uneventful. Gave physostigmin (eserin) to use constantly.

November 17: Vision, right, 3/200; left, 18/200; tension + 1.

June 24: Vision, right, 20/200; left, 12/200; tonometer, right, 65 mm. Hg; left, 32 mm. Hg (physostigmin).

July 2: No miotic, tonometer, right, 69 mm. Hg; left, 38 mm. Hg; fundus, each eye, marked cupping with atrophic nerves. Has developed tuberculosis and has been sent to county sanatorium.

November 4: Right, 2/200 — 1.50 ○ — 0.50 × 60 = 20/200; left, 20/200 — 1.25 ○ — 1.50 × 165 = 20/70; tonometer, right, 75 mm. Hg; left, 60 mm. Hg. (no miotic). Chambers very

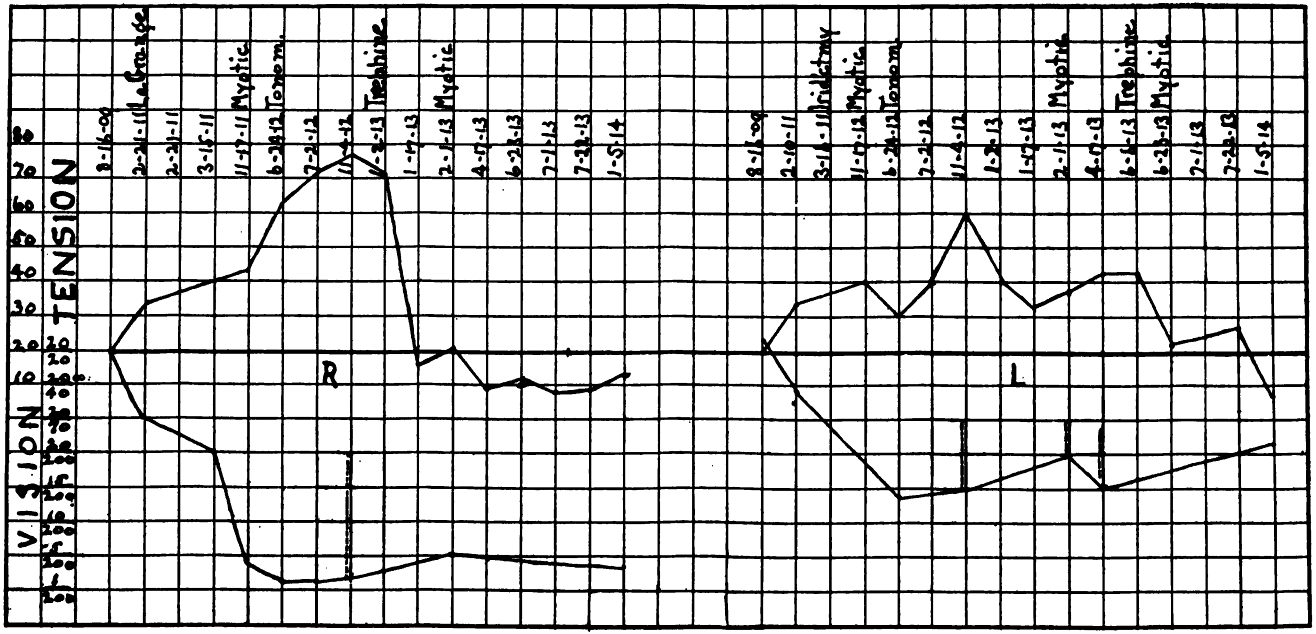

Fig. 2.—Vision and tension in Case 3. Dotted lines indicate improvement with glasses.

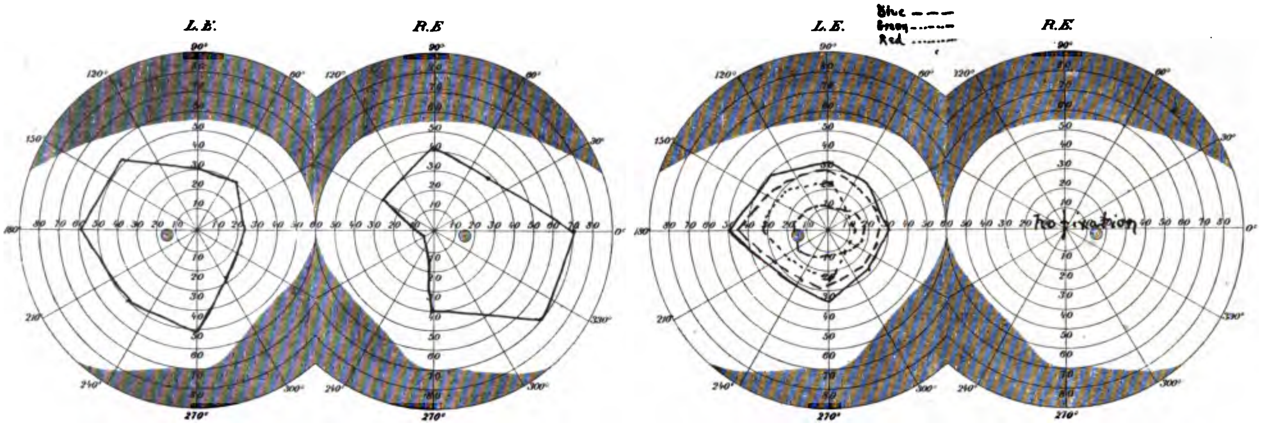

Fig. 3.—Field of vision in Case 3, taken Sept. 11, 1911, and Nov. 3, 1912. Confusion of colors indicated by crossing of colors.

deep. Atrophic nerves, with cupping. Calls green light blue; left field much contracted, health much better.

Jan. 2, 1913: Tonometer, right, 70 mm. Hg; left, 40 mm. Hg. Operation; right eye, scleral trephine.

January 3: Chamber restored.

January 4: Eye quiet.

January 17: Tonometer, right, 15 mm. Hg; left, 33 mm. Hg.

February 7: Right 6/200, unimproved; left, 20/200 — 1.25 ⊃ + 2×165 = 20/70; tonometer, right, 20 mm. Hg; left, 37 mm. Hg (physostigmin).

April 17: Tonometer, right, 8 mm. Hg; left, 44 mm. (physostigmin).

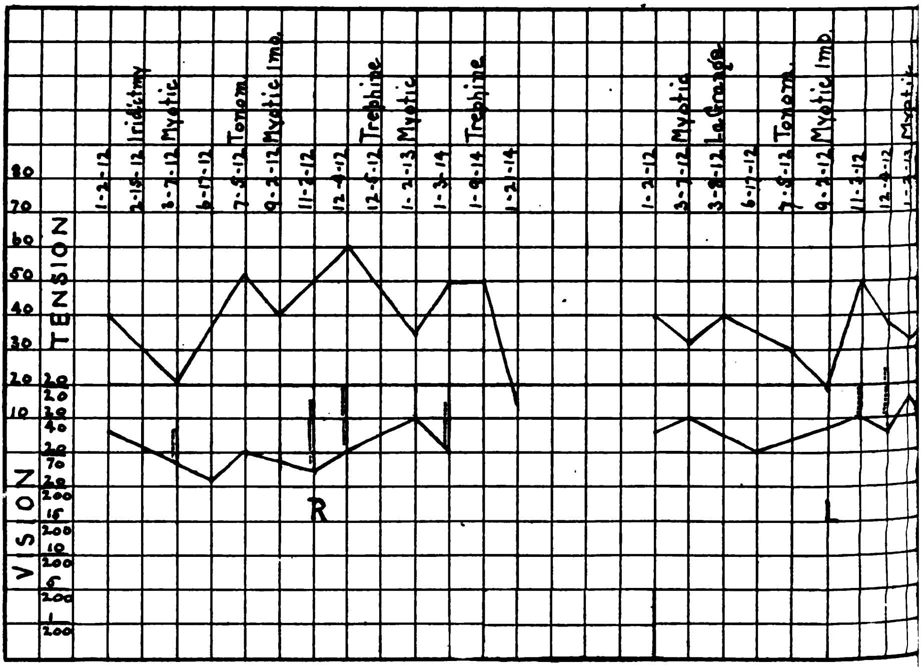

Fig. 4.—Vision and tension in Case 4. Dotted lines indicate improvement with glasses.

April 18: Right, 5/200; left 15/200; with glass = 20/100.

June 6: Operation: Left, scleral trephine.

June 7: Chamber restored.

June 9: Comfortable.

June 23: Tonometer, right, 11 mm. Hg; left, 23 mm. Hg (physostigmin).

July 1: Tonometer, right, 7 mm. Hg; left, 25 mm. Hg.

July 22: Tonometer, right, 7 mm. Hg; left, 27 mm. Hg.

Jan. 5, 1914: Vision, right, 4/200; left, 20/100, unimproved; tonometer, right, 14 mm. Hg; left, 7 mm. Hg; good filtration scars.

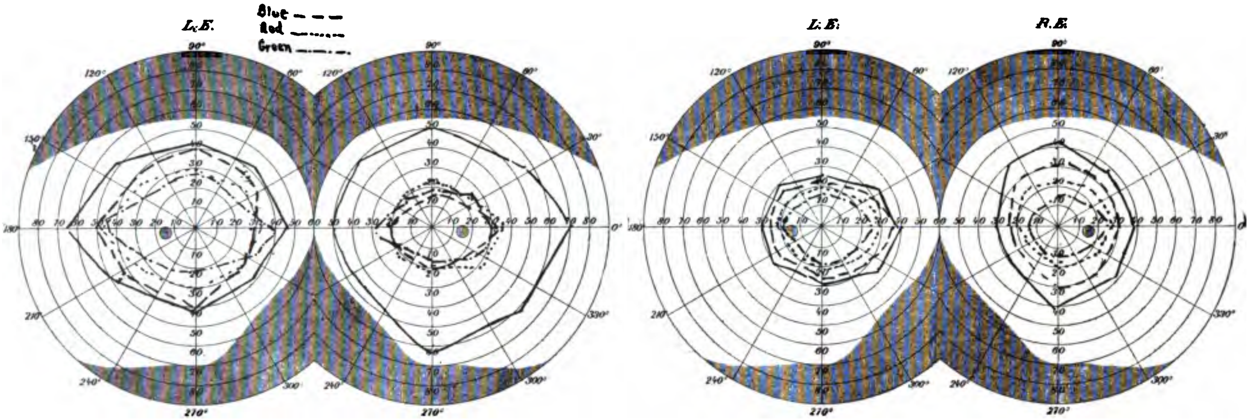

Fig. 5.—Fields of vision in Case 4, first taken Jan. 2, 1912, before miotic and later, after use of miotic.

Case 4.—E. G., second son of Patient 2, aged 16, was first seen Jan. 2, 1912. His health has always been good, but sight has been failing three months; no other symptom of eye trouble is present. Examination: Right eye, vision 20/50 unimproved; anterior chamber deep; fundus normal; tension + 1; field, slightly contracted for form, markedly for colors. Left eye, 20/50 unimproved; rest of examination same as right; field more contracted. Irises brown, diameter of cornea 11.5 mm.

February 16: Iridectomy (right) section made with knife; patient allowed to leave operating-room for home.

February 16: Anterior chamber unrestored.

February 17: Chamber slowly restoring.

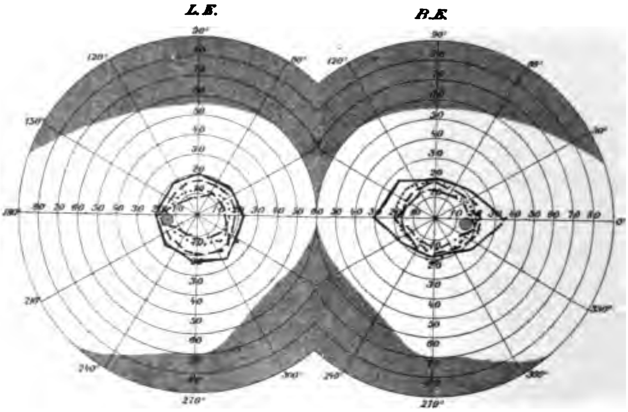

Fig. 6.—Fields of vision in Case 4 nearly 12 months later. No miotics, and great contraction for form.

March 7: Vision, right, 20/70 + 0.50 × 45 = 20/50; tension (digital) normal. Vision, left, 20/40 (physostigmin). (Vision and tension shown in Fig. 4.)

March 8: La Grange operation (left); patient allowed to go home.

March 10: Chamber unrestored.

March 11: Chamber slowly restoring.

June 17: Right eye, 20/100. Left eye, 20/70.

July 5: Tonometer, right, 53 mm. Hg; left, 34 mm. Hg; vision, right, 20/70; left, 20/50 using physostigmin one month.

September 2: Tonometer, right, 40 mm. Hg; left, 18 mm. Hg (physostigmin).

November 3: Right, 20/100 — 1 × 120 = 20/30; left, 20/40 + 0.50 ⊃ — 1 × 75 = 20/20; field very contracted. Tonometer, right and left, 50 mm. Hg.

December 4: Right, 20/70 + 0.50 ⊃ — 1.50 × 105 = 20/20; left, 20/50 — 1 × 75 = 20/15; tonometer, right, 60 mm. Hg; left, 38 mm. Hg.

December 5: Operation: Right, scleral trephine; large conjunctival flap containing much scar-tissue. Trephine not a perfect one, although good scleral button removed.

December 6: Chamber restored.

December 11: Chemosis.

December 18: Eye quiet.

December 28: Eye white.

Jan. 3, 1914: Right, 20/70 — 50 ⊂ + 1.50 X 30 = 20/30. Left, 20/40 + 0.75 X 165 = 20/15. Tonometer, right, 52 mm. Hg; left, 36 mm. Hg. No filtration scar in right eye. Operation advised.

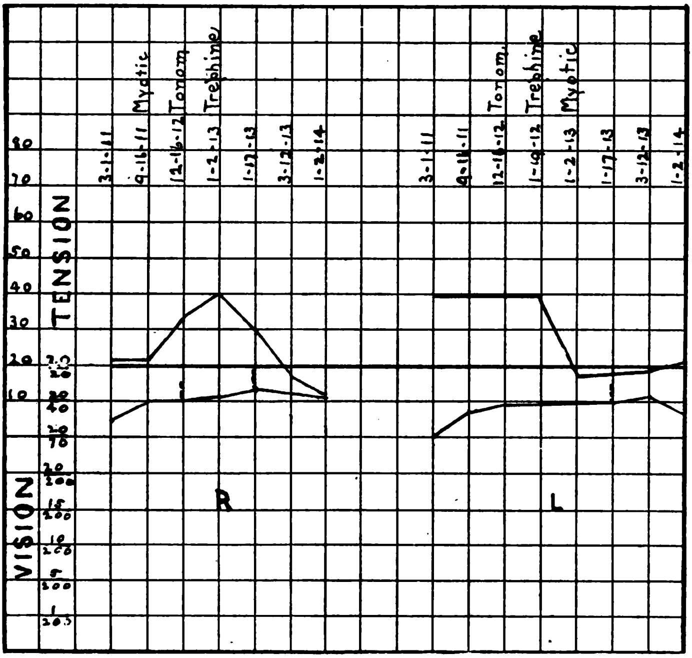

Fig. 7.—Vision and tension in Case 5. Dotted lines indicate improvement with glasses.

January 9: Operation: Scleral trephine right eye, corresponding to 1 o'clock on watch dial. Not perfectly done on account of scar tissue of former operations, although good button of sclera removed.

January 21: Tonometer, right, 13 mm. Hg; good filtration scar and eye very quiet.

Case 5.—B. G., aged 13, third son of Patient 2, seen March 1, 1911. His health is good; sight has been failing for some months. Examination: Right 20/50 unimproved. Tension

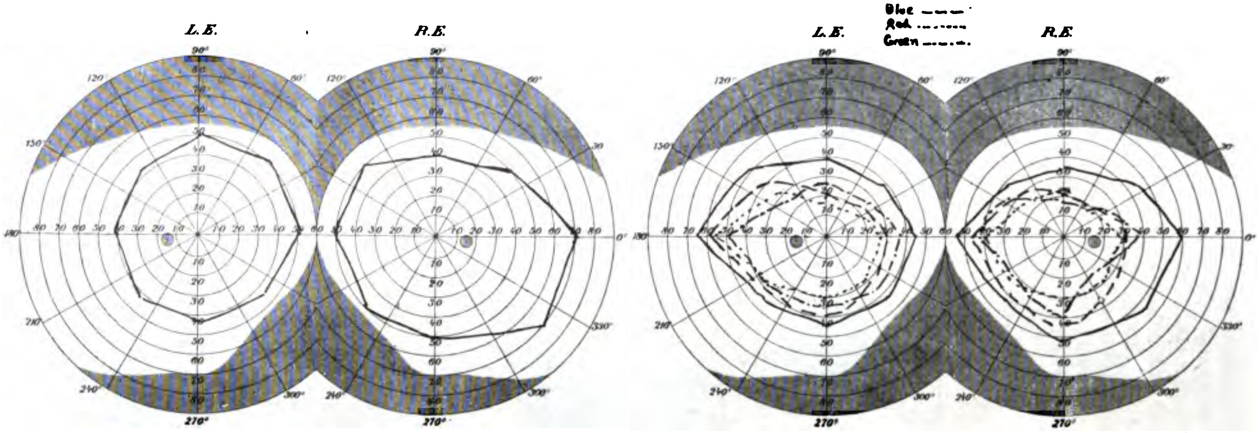

Fig. 8.—Fields of vision in Case 5, taken March, 1911, and Dec. 17, 1912. Great confusion of colors.

normal, rigidity of sclera; field contracted for form, great confusion of colors; fundus normal, except pale nerve; anterior chamber deep. Left, 20/70 unimproved. Tension +1; field very contracted and great confusion of colors. Diameter of cornea, right, 11.5 mm.; left, 12 mm.; irises light brown. Gave physostigmin.

September 16: Vision, right, 20/40; vision, left, 20/50; tension same. (Vision and tension shown in Fig. 7.)

September 2: Developed tuberculosis.

December 16: Right, 20/40 + 37 × 15 = 20/30; left, 20/40 + 0.50 × 105 = 20/40. Chambers appear of normal depths. Tonometer, right, 35 mm. Hg; left, 40 mm. Hg. Fundus, right, pallor to temporal half of disk, pulsation of large vein; left, decided pallor to temporal half of disk with atrophic chorioid adjoining temporal side of disk.

December 19: Operation: left, scleral trephine with iridectomy. Allowed to go home.

December 23: Unrestored chamber; pain in eye.

December 24: Chamber restored.

December 28: Eye quiet.

January 2: Tonometer, right, 40 mm. Hg.; left, 14 mm. Hg. Operation: right, scleral trephine with iridectomy. Allowed to go home.

January 7: Chamber restored slowly.

January 17: Right, 20/30 + 25 × 45 = 20/20; left, 20/40 + 50 × 15 = 20/30.

March 12: Tonometer, right, 15 mm. Hg; left, 18 mm. Hg. Scope, one vessel, cupping in right disk. Good filtration scars.

January 2: Right, 20/40 + 0.50 × 90 = 20/40; left 20/50 — 50 × 180 = 20/50. Tonometer, right, 14 mm. Hg; left, 23 mm. Hg; flat filtration scars; fundus, no change; condition satisfactory.

Case 6.—P. H., aged 39, sister of patient in Case 2. Past history unimportant, malaria several times. Attention called to eyes at 11 years of age on account of inability to read well. Always saw "rings" around lights. Left eye inflamed at 3 years of age and has never been able to see. At the age of 12, right eye operated on and sight since has been good. Examination: Right, 20/70, unimproved. Tension: Tonometer, 17 mm. Hg; chamber deep; scope optic atrophy; little cupping; vessels of normal caliber. Good broad base iridectomy. Field very contracted. Left aphakic eye. Iridodonesis, optic atrophy, cupping of vessels. Diameter of cornea, 11 mm.; blue irises.

Case 7.—L. S., aged 14, came to the clinic to have his eyes examined, March 3, 1911, as all of his cousins and most of his family had bad sight. He was unaware that his sight was defective. He too, like other members of this series, had malaria. Examination: Right, 20/70, very deep chamber; fundus normal; hyperopia 2 D; field, some peripheral contraction. Left, 20/20, deep chamber; fundus, normal; field, some contraction; hyperopia, 1 D. Blue irises, tension normal (digital) but rigid sclera; diameter of corneas 12 mm. I did not consider that he had glaucoma, but he was directed to return for further observations.

February 12: Right, 20/70; left, 20/40; fields, decided narrowing with great confusion of colors, so much so that accurate charts could not be made. Patient given physostigmin.

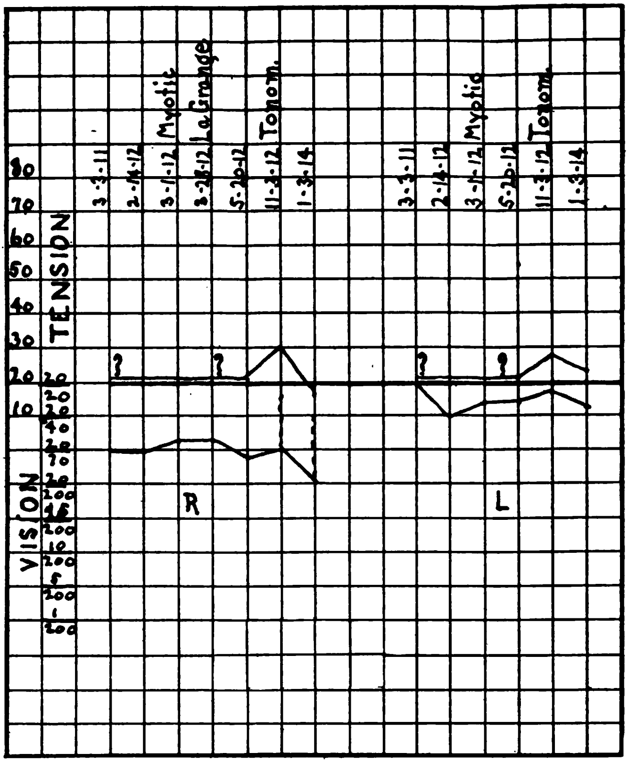

Fig. 9.—Vision and tension in Case 7. Dotted lines indicate improvement with glasses.

March 1, 1912: Right, 20/50; left, 20/30; Tension normal March 15, fields, decided contraction in right eye. (Vision and tension shown in Fig. 9.)

March 28: Operation: Right, LaGrange sclerectomy with iridectomy. Allowed to return home.

March 29: Chamber restored, very deep.

May 20: No physostigmin, right, 20/70; left, 20/30.

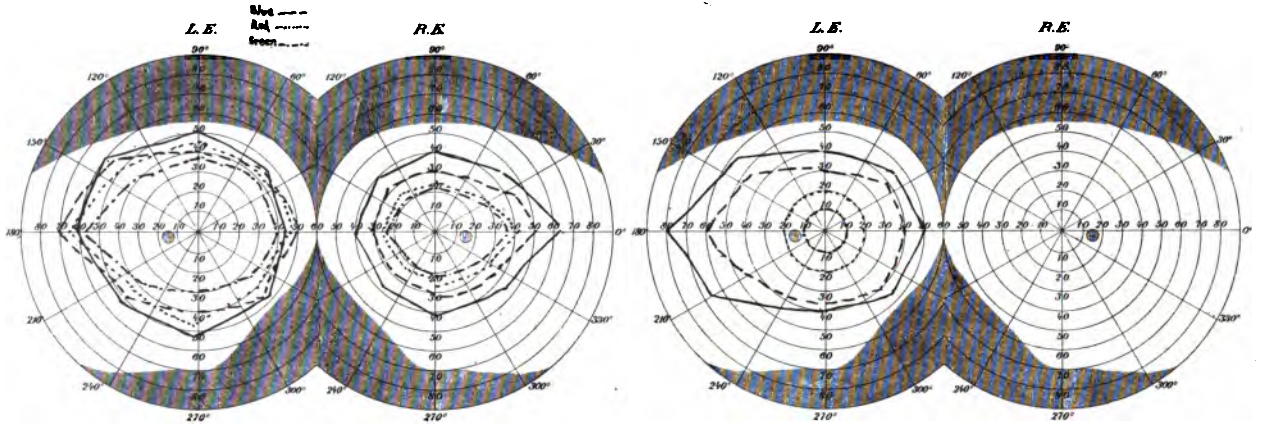

Fig. 10.—Fields of vision in Case 7, taken March 15, 1912. The contraction of the field in the right eye, and the confusion of colors in the left eye, led to operation on the right eye. The second field was taken for the left eye only, Jan. 3, 1914.

November 3: Fundus, each eye, vessels normal, pale nerves, tonometer with difficulty, 27 mm. Hg; fields normal, Right, 20/70 — 1.50 S = 20/30; left, 20/20 — 0.50 × 90 = 20/20; patient lost sight of for several months.

Jan. 3, 1914, right, 20/200 — 1.50 S = 20/30; left, 20/30 + 50 × 90 = 20/30. Tonometer: right, 15 mm. Hg; left, 25 mm. Hg. Field, left decided contraction for red and green. Fundus right, slightly pale nerve, left normal. Anterior chamber very deep and the light reflex on the cornea appears like that in keratoconus. Even now I am not positive that this patient has glaucoma, but with a variable vision, a contracted field for form and colors, and with the unquestionable family history, he should be regarded as "suspicious." I believe the sclerectomy performed has been of help.

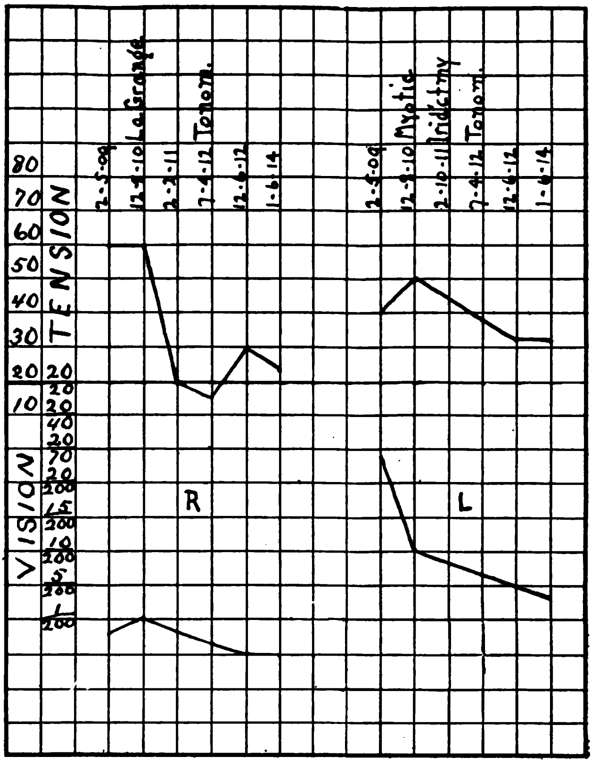

Fig. 11.—Vision and tension in Case 8.

CASE 8.—C. R. H., aged 23, brother of Patients 2 and 3. His past history is negative. Five months before the first visit, Feb. 5, 1909, he noticed that his sight was failing. Examination: Right, light perception; atrophic nerve; contracted vessels and marked cupping; chamber very deep. Tension +2. Left, 20/70, deep chamber; atrophic nerve; cupping of vessels, which were moderately contracted. Tension + 1; field, marked contraction of nasal half. Brown irises, diameter of corneas 12 mm. Hg. Gave physostigmin.

December 8 to 10: Seen for the first time in several months. Has been using drops almost continually. Right, peripheral vision only. Left, 10/200. Operation: Right eye, LaGrange sclerectomy, with iridectomy. Chamber not restored for four days. Kept in hospital bed, very quiet. Result very good at dismissal.

Feb. 2, 1911: Tension, normal right (digital). (Vision and tension shown in Fig. 11.)

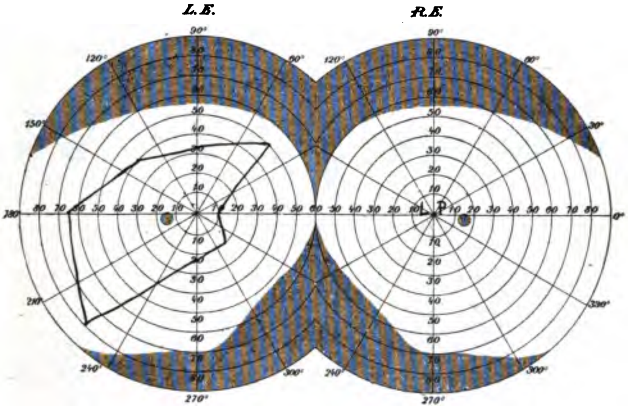

Fig. 12.—Fields of vision in Case 8, taken late in the disease.

February 10: Operation: Left iridectomy; allowed to leave hospital. Chamber restored in two days.

July 4, 1912: Tonometer, right, 18 mm. Hg; left, 38 mm. Hg (no physostigmin). Fundus no change.

December 6: Right, light perception; left, 5/200. Tonometer: right, 29 mm. Hg; left, 34 mm. Hg.

Jan. 6, 1914: Right, light perception; left, 4/200. Tonometer: right, 24 mm. Hg; left, 33 mm. Hg. Fundus, optic atrophy complete, vessels thread-like.

LAWFORD'S CASE.—McL., blind for some years before death. Information from two daughters, undoubtedly chronic glaucoma. He had thirteen children, of which the first, third and seventh died in infancy; the fifth and ninth from accidents and paralysis. The sixth became attacked with bilateral glaucoma when 66 and was operated on by Lawford. She died at 71 from cerebral hemorrhage. She had seven children, all living. Author has seen all but the first. Three of them, the second, third and fifth are affected. The third and fifth have been operated on for marked chronic glaucoma, coming on at the age of 48 and 40 respectively. The second child has no objective symptoms (doubtful rainbows once), but showed slight contraction of the left field in its basal part and a large deep cup in left disk. In Generation IV there are eleven persons, children of the first and second offspring in Generation III. The eldest in the first family is 27 years of age; the eldest in the second family 29; all are reported to have good sight. "Anticipation" is well exemplified in this (For pedigree see Fig. 13).

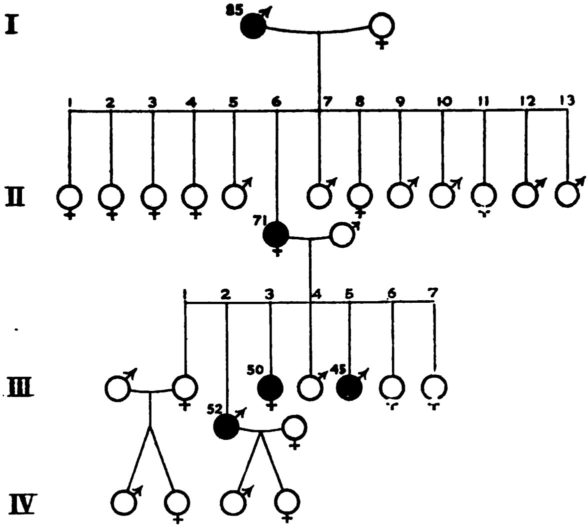

Fig. 13.—Pedigree of Lawford's patient.

Lucien Howe's Case.—"Anticipation" was well pronounced in this family. In Generation I, the fourth born developed glaucoma at 40 which led to complete blindness. The two affected children, in Generation II, the second and fourth, developed the disease at 28 and 25 respectively. In Generation III, the age of onset in the five persons attacked varied from 28 to 17 (Pedigree given in Fig. 14).

Nettleship's Case.—Three persons in three generations were affected and were all seen and operated on by the author. In Generation I, the disease began about the age of 71; in Generation II about the age of 40 and 48, and in Generation III between the ages of 25 and 30. A cousin of Generation II, the third on the maternal side is said to have been operated on for glaucoma (Pedigree given in Fig. 15).

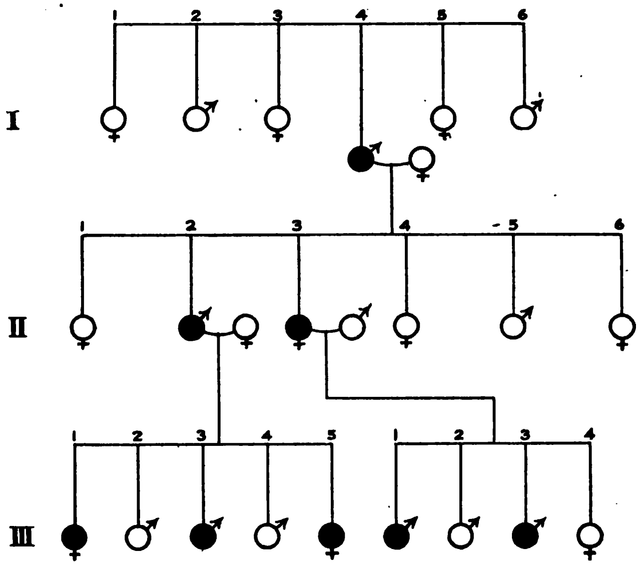

Fig. 14.—Pedigree of Howe's patient.

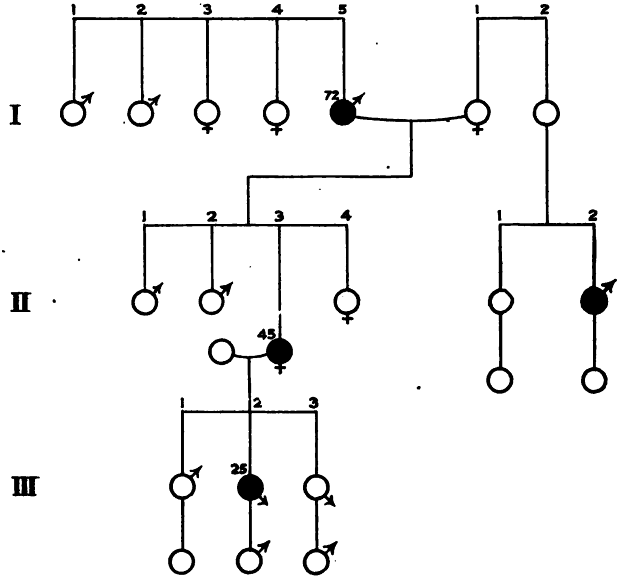

Fig. 15.—Pedigree of Nettleship's patient.

**Rudin's Case.**—Generation I. Female blind from glaucoma at 30. Generation II, son of Generation I, became blind at 30. Generation III, two sons of Generation II were affected at 27 and 20 respectively.

In the review of the cases of glaucoma simplex of two and three generations, various comments by many authors were noted. Benedict in his early work cites a history of a gouty family all affected with glaucoma except one son, and he escaped because he had blue eyes. Arlt and Fischer, too, mention this phenomena, that in eyes with blue or gray irises glaucoma rarely develops.

Rogman reports a case of acute glaucoma developing from glaucoma simplex. Physiologic cupping was noted in the unaffected eye of one of the sons. The same condition with physiologic cupping was detected in a second son, who subsequently developed acute glaucoma. A third son has a similar picture of physiologic cupping in each eye with a hyperopia of 2.50 D. In none of these cases was there typical glaucomatous cupping, yet there was always a decided elevation in tension and diminished vision.

Priestley Smith has reported notes on glaucoma in two generations. A father, aged 65, hyperopia, 2.50 D.; horizontal diameter of the cornea, 10.5 mm.; a daughter, aged 29, left eye, hyperopia, 6 D. The right eye was excised for absolute glaucoma, and the measurements were: horizontal diameter of the cornea, 10 mm.; globe horizontally, 22 mm.; vertically, 21 mm.; pole to pole, 21.75 mm. The normal horizontal diameter of cornea is between 11 and 12 mm., and the same author ventures the assertion that an eye with a cornea whose horizontal diameter is 10.5 mm. rarely escapes glaucoma. Glaucoma simplex of the hereditary type developing in myopic eyes is conspicuously mentioned, especially in Somya's report of a family.

## SUMMARY

In summarizing the reported cases of hereditary glaucoma (simplex) we may briefly say that:

1. "Anticipation" is the most prominent feature, as Lawford mentions. Hereditary glaucoma, usually, I might say always, develops in adults, or at an age far remote from the usual period, and whenever a case of

glaucoma simplex is recognized in one under the age of 30, interest should be aroused as to the hereditary tendency.

2. The smallness of the cornea and globe does play an important part; it however, is not the sole cause, for two families of myopes have been reported. Unfortunately few corneas have been measured.

3. General diseases other than gout and rheumatism, mentioned by the older writers, have small part in the causation of this disease.

4. The liability to transmission by the two sexes is roughly equal. The male sex shows a greater liability to inheritance.

A study of the family which I have reported shows the remarkable fact that defective vision was discovered at an early age in all cases. The father, when a young man of 30, unquestionably had glaucoma, and an iridectomy was performed. The youngest age was 11 (Case 6) and the sister (Case 2) first noticed dim sight at the age of. 12, whereas in the cases of her three sons, (Cases 3, 4 and 5) they were some years older. The normal refraction of the eyes was emmetropic, or slightly hypermetropic. After operations the corneal astigmatism frequently changed to minus or mixed astigmatism.

In five of the cases, thirteen operations were performed on nine eyes. The right eye in Case 4 received an iridectomy and two trephines, until lowered tension was obtained. In my hands the iridectomy as performed for glaucoma and the sclerectomy with iridectomy of LaGrange, did not give the results which were finally obtained by the scleral trephine. Time will tell, however, whether or not the subnormal tension of 7, 11 mm. Hg thus obtained acts detrimentally to the glaucomatous eye.

As for the results in the five of the eight cases which I personally cared for, we might reasonably say that after atrophic change and cupping had taken place in the nerve (Cases 3 and 8), the operations have prolonged sight and possibly arrested in a measure early blindness, whereas in the early cases without ophthalmoscopic nerve-changes, with only a reduction of vision, elevation in tension and contracted fields as symptoms of the disease, the results are gratifying. In this connection attention should be directed to the fact that after all of the thirteen operations except two (Case 8), the patients were immediately allowed to return to their homes, and no complication, unusual delay in healing or bad after-effect was the result.

### CONCLUSIONS

1. The sizes of the corneas were within normal limits.

2. The anterior chambers were all of normal depth and the pupillary reactions were sluggish.

3. While the visual fields were not characteristic, there uniformly existed a great confusion of colors.

4. A physiologic cupping was noted as an early symptom in two cases.

5. Many of the irises were of light color, a fact of no importance.

6. The earliest symptom was a diminution of vision in one eye.

7. Miotics were always useful and even after operations they aided in the reduction of tension.

8. The tonometer is a most valuable and necessary instrument in noting the progress of the disease, as much as the clinical thermometer in any febrile condition.

9. The operation of choice in my hands, the one that maintains low intra-ocular tension, is the scleral trephine after the manner of Elliot.

10. The cause of this form of glaucoma as well as the part that heredity plays is still unknown. While, unfortunately, no microscopic studies have yet been made, we can reasonably assume that some congenital obliteration or sclerosis exists in the region of Schlemm's canal or changes similar to those which occur in the buphthalmic eye.

11. A sociological question arises whether the children of such a glaucomatous family should marry and become parents.

Candler Building.

# MEMORANDA

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# SCLEROCORNEAL TREPHINING FOR GLAUCOMA *

WALTER R. PARKER, M.D.
DETROIT

## I. REPORT OF COLONEL ELLIOT'S CASES

At the request of Chairman Todd, and with the consent of Colonel Elliot, I have the privilege of presenting to the members of the Section the results of the sclerocorneal trephining operations for the relief of glaucoma performed by Col. R. H. Elliot during his visit to the United States last fall.

TABLE 1.—SURGEONS REPORTING OPERATIONS BY COLONEL ELLIOT

| Name | No. of Operations |
|---|---|
| Robert Beattie | 1 |
| F. E. Cheney | 1 |
| A. E. Davis | 2 |
| L. Webster Fox | 10 |
| W. H. Fox | 1 |
| P. J. Pontius | 1 |
| George S. Derby | 1 |
| J. M. Ray | 2 |
| R. A. Reeve | 5 |
| G. C. Savage | 3 |
| Frank C. Todd | 18 |
| E. Torok | 1 |
| W. H. Wendell | 2 |
| W. H. Wilmer | 3 |
| E. B. Cayce | 1 |
| L. W. Callan | 2 |
| E. T. Easton | 2 |
| John Green, Jr. | 4 |
| A. G. Morse | 1 |
| W. R. Parker | 6 |
| Samuel D. Risley | 1 |
| McCluney Radcliffe | 2 |
| William M. Sweet | 1 |
| George E. deSchweinitz | 2 |
| H. H. Tyson | 1 |
| Will Walter | 1 |
| F. E. Woodruff | 1 |
| Total | 76 |

In preparing this report, circular letters were sent out to those for whom Colonel Elliot operated, and

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

* A report of the tabulated results of seventy-six operations performed by Col. R. H. Elliot during his visit to the United States, and a clinical study of twenty-seven operations by the author.

reports of cases were received from the surgeons named in Table 1, to whom I wish to express my sincere appreciation for their hearty cooperation.

TABLE 2.—SUMMARY OF OPERATIONS BY COLONEL ELLIOT

| | |
|---|---|
| Patients operated on | 61 |
| Eyes operated on | 76 |

| | No. | Results Good | Results Poor | Results Failure |
|---|---|---|---|---|
| Males, right eye | 9 | 7 | 0 | 2 |
| Males, left eye | 9 | 7 | 1 | 1 |
| Total | 18 | 14 | 1 | 3 |
| Females, right eye | 16 | 11 | 2 | 3 |
| Females, left eye | 12 | 11 | 0 | 1 |
| Total | 28 | 22 | 2 | 4 |
| Males, both eyes | 8 | 15 | 1 | 0 |
| Females, both eyes | 7 | 11 | 1 | 2 |
| Total | 15 | 26 | 2 | 2 |
| Eyes trephined after iridectomy and glaucoma extraction | 11 | 10 | 1 | 0 |
| Eyes trephined after iridectomy for cataract | 4 | 2 | 0 | 2 |
| Total | 15 | 12 | 1 | 2 |

Good results trephined after iridectomy ................. 90.9 per cent.
Good results trephined after combined extraction of cataract ................................. 50 per cent.

TABLE 3.—NUMBER OF CASES, TYPES OF GLAUCOMA AND RESULTS IN EACH GROUP OF CASES

| Type of Glaucoma | | Previous Iridectomy | Without Iridectomy | Complete Iridectomy | Partial Iridectomy | Iritis | Without Iritis | Results Good | Results Poor | Results Failure |
|---|---|---|---|---|---|---|---|---|---|---|
| Absolute | 1 | 1 | .. | .. | .. | .. | .. | .. | .. | 1 |
| Acute inflammatory | 7 | .. | .. | 1 | 6 | .. | 5 | 6 | .. | 1 |
| Simple | 61 | 8 | 8 | 6 | 39 | 26 | 4 | 51 | 5 | 5 |
| Secondary | 5 | 4 | .. | .. | 1 | 1 | 4 | 4 | .. | 1 |
| Hemorrhagic | 2 | .. | 1 | .. | 1 | 1 | 1 | 1 | 1 | .. |
| | 76 | 13 | 9 | 7 | 47 | .. | .. | .. | .. | .. |

GOOD RESULTS IN DIFFERENT TYPES OF GLAUCOMA

| | No. of Cases | Per Cent. |
|---|---|---|
| Simple glaucoma | 61 | 83.6 |
| Acute inflammatory | 7 | 85.7 |
| Secondary | 5 | 80 |
| Hemorrhagic | 2 | 50 |
| All cases except simple | 15 | 73.3 |

The classification of results in Table 4 is based on the effect the operation produced on the tension of the eyeball, the data concerning the acuity of vision before and after operation being too meager to justify conclusions.

### TABLE 4.—RESULTS OF TREPHINING

| | No. | Per Cent. |
|---|---|---|
| Operations | 76 | ..... |
| Good results | 62 | 81.58 |

| | No. | Good | Poor | Failure | Not Stated |
|---|---|---|---|---|---|
| Eyes having had previous iridectomy | 13 | 11 | 1 | 1 | 0 |
| Eyes having complete iridectomy at time of trephining | 7 | 7 | 0 | 0 | 0 |
| Total | 20 | 18 | 1 | 1 | 0 |
| Good results in 90 per cent. | | | | | |
| Eyes having partial iridectomy at time of trephining | 47 | 37 | 5 | 5 | 0 |
| Eye in which no iridectomy was done | 9 | 7 | 0 | 2 | 0 |
| Total | 56 | 44 | 5 | 7 | 0 |
| Good results in 78.4 per cent. | | | | | |

### TABLE 5.—CLASSIFICATION WITH REFERENCE TO THE PRESENCE OR ABSENCE OF IRITIS IN CASES IN WHICH A PREVIOUS IRIDECTOMY HAD BEEN PERFORMED OR A COMPLETE IRIDECTOMY WAS DONE AT THE TIME OF OPERATION

TREPHININGS FOLLOWED BY IRITIS

| | No. | Iritis | No Iritis | Not Stated |
|---|---|---|---|---|
| Cases having complete iridectomy at time of trephining | 7 | 1 | 5 | 1 |
| Having had previous iridectomy | 13 | 1 | 10 | 2 |
| Total | 20 | 2=10% | 15=75% | 3 |
| Having no iridectomy | 9 | 7 | 2 | 0 |
| Having partial iridectomy | 47 | 22 | 23 | 2 |
| Total | 56 | 29=51% | 25=44.6% | 2 |

Of the twenty cases in which an iridectomy had been made, either at time of operation or previously, 10 per cent. showed signs of iritis, whereas in the fifty-six cases in which the trephining had been performed without or with a partial iridectomy, iritis was present in 51.7 per cent.

I have to acknowledge my great indebtedness to Colonel Elliot for his kindness in allowing me to present the results of his splendid work before the members of this Section.

My thanks are due to my assistant, Dr. William L. Benedict, for valuable assistance in tabulating the statistics here presented.

## II. REPORT OF AUTHOR'S CASES

In the twenty-seven cases here reported, operation was performed at the Ophthalmic Clinic of the University of Michigan, except two, which were done in private practice.

I shall not discuss the technic of the sclerocorneal trephining operation, but rather confine my report to the clinical facts as presented by the case histories, and base my conclusions on these findings alone.

TABLE 6.—SUMMARY OF AUTHOR'S CASES

| | | | |
|---|---|---|---|
| Males | 13 | Eyes operated on | 17 |
| Females | 7 | Eyes operated on | 10 |
| | 20 | | 27 |

| | |
|---|---|
| Cases in which both eyes were trephined | 5 |
| Cases in which the same eye was trephined twice | 2 |

TABLE 8.—NUMBER AND TYPES OF CASES, TOGETHER WITH THE RESULTS OBTAINED IN EACH TYPE OF GLAUCOMA

| Classification | No. of Cases | Good No. % | Poor Results No. | Failure No. |
|---|---|---|---|---|
| Acute | 1 | .. | .. | 1 |
| Simple | 14 | 11=78.5 | 2 | 1 |
| Chronic inflammatory | 6 | 4=66.6 | 2 | .. |
| Secondary to cataract extract | 2 | .. | 1 | 1 |
| Secondary to luxated lens | 1 | .. | .. | 1 |
| Secondary to traumatism | 1 | 1 | .. | .. |
| Buphthalmos | 2 | .. | .. | 2 |
| | 27 | 16 | 5 | 6 |

TABLE 9.—RESULTS OBTAINED IN CASES OF SIMPLE GLAUCOMA, COMPARED WITH THOSE IN ALL OTHER TYPES OF THE DISEASE AS CLASSIFIED

| Type | No. | Results<br>Good<br>No. % | <br>Poor<br>No. % | <br>Failure<br>No. % |
|---|---|---|---|---|
| Simple | 14 | 11=78.5 | 2=14.2 | 1= 7.1 |
| Other types | 13 | 5=38.4 | 3=23.0 | 5=38.4 |
| | 27 | 16 | 5 | 6 |

Good results from simple cases.............78.5 per cent.
Good results from all other cases...........38.5 per cent.

TABLE 10.—CLASSIFICATION WITH REFERENCE TO THE PRESENCE OR ABSENCE OF IRITIS IN CASES IN WHICH A PREVIOUS IRIDECTOMY HAD BEEN PERFORMED OR A COMPLETE IRIDECTOMY WAS DONE AT THE TIME OF OPERATION

| Character of Iridectomy | No. | Iritis No. Per Ct. | No Iritis No. Per Ct. |
|---|---|---|---|
| Complete iridectomy at time of trephine operation | 8 | 1 = 12.5 | 7 = 87.5 |
| Previous iridectomy | 7 | 2 = 28.5 | 5 = 71.4 |
| Partial iridectomy | 8 | 6 = 75.0 | 2 = 25 |
| Without iridectomy | 4 | 4 = 100 | |
| | 27 | 13 | 14 |

## TABLE 7.—VISION AND TENSION BEFORE AND AFTER OPERATION, AND RESULT AS DETERMINED BY TENSION AT TIME OF LAST OBSERVATION

| Case No. | Date | Type | Vision Before—After Operation * |  | Tension Before mm. | Tension Date | Tension After mm. | Result | Remarks |
|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Before | After |  |  |  |  |  |
| 1 | 12/ 9/12 | Traumatic secondary. | Nil | Nil | 95 | 4/ 9/13 | 21 | Good |  |
| 2 | 12/16/12 | Simple | 6/15 | 6/15 | 44 | 2/ 2/13 | 21 | Good |  |
| 3 | 5/ 3/13 | Buphthalmos | ? | ? | 48 | 7/14/13 | 70 | Failure |  |
| 4 | 5/ 3/13 | Buphthalmos | ? | ? | 48 | 7/14/13 | 60 | Failure |  |
| 5 | 5/26/13 | Simple | Nil | Nil | 70 | 6/ 4/13 | 16 | Good |  |
| 6 | 5/26/13 | Secondary cataract | 5/30 | 1/60 | 83 | 7/12/13 | 47 | Poor | Immediate result good 17 days after operation. Tension 16 mm. Discharged cured. One month later, tension 47. |
| 7 | 7/ 7/13 | Simple | 1/60 | l. p. | 47 | 7/ 8/13 | 20 | Good |  |
| 8 | 10/31/12 | Chronic inflammation. | Nil | Nil | 60 | 3/13/13 | 13 | Good |  |
| 9 | 10/3 /12 | Chronic inflammation. | Nil | Nil | 71 | 3/13/13 | 18 | Good |  |
| 10 | 10/10/12 | Simple | l. p. | l. p. | 39 | 1/27/14 | 16 | Good |  |
| 11 | 10/17/12 | Simple | l. p. | l. p. | 105 | 3/24/14 | 10 | Good |  |
| 12 | 4/24/13 | Simple | 5/60 | 1/60 | 45 | 3/24/14 | 27 | Good |  |
| 13 | 10/17/12 | Simple | Obj. | Obj. | 60 | 10/ 3/12 | 12 | Good |  |
| 14 | 10/31/12 | Secondary | l. p. | Nil | 36 | 2/ 7/13 | 100 | Failure | Immediate results good. Tension at end of two weeks 14 mm. |
| 15 | 11/21/12 | Chronic inflammation. | Nil | Nil | 83 | 12/22/12 | 82 | Failure | Hemorrhagic, Glau. O. S. |
| 16 | 2/24/13 | Chronic inflammation. | Nil | Nil | 60 | 5/ 5/13 | 60 | Failure |  |
| 17 | 6/30/13 | Chronic inflammation. | Nil | Nil | 60 | 3/ 5/14 | 20 | Good |  |
| 18 | 4/24/13 | Sec. luxated lens | Nil | Nil | 91 | 1/ 7/14 | 55 | Failure |  |
| 19 | 7/ 7/13 | Simple | 5/60 | 5/60 | 51 | 11/ 6/13 | 15 | Good |  |
| 20 | 7/21/13 | Simple | 5/60 | 6/60 | 51 | 11/ 6/13 | 7 | Good |  |
| 21 | 11/20/13 | Chronic inflammation. | Fingers | 6/30 | 50 | 1/19/14 | 18 | Good |  |
| 22 | 10/20/13 | Simple | Nil | Nil | 60 | 10/31/13 | 70 | Poor |  |
| 23 | 10/10/13 | Simple | Nil | Nil | 44 | 10/31/13 | 60 | Poor |  |
| 24 | 1/ 8/14 | Acute inflammation | l. p. | Nil | 62 | 2/ 2/14 | 50 | Poor | Immediate results good. Tension 24, ten days after operation. |
| 25 | 1/12/14 | Simple | Nil | Nil | 73 | 3/19/14 | 36 | Good | Subsequent to last tension record eye soft, but tonometer record not recorded. |
| 26 | 1/14/14 | Simple | 6/20 | 6/20 | 36 | 2/ 4/14 | 26 | Good |  |
| 27 | 11/20/13 | Simple | 6/9 | Obj. | 43 | 4/ 6/14 | 15 | Poor | Immediate result perfect, chamber closed on 19th day. Quiet iritis, with exudate in pupillary space. |

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* In this column l. p. stands for light perception.

TABLE 11.—FREQUENCY OF OCCURRENCE OF IRITIS IN THE DIFFERENT FORMS OF IRIDECTOMY

| | | Iritis | No Iritis |
|---|---|---|---|
| | No. | No. Per Ct. | No. Per Ct. |
| Total number of cases of complete and previous iridectomy .............. | 15 | 3 = 20 | 12 = 80 |
| Total number of cases of partial or without iridectomy ............... | 12 | 10 = 83 | 2 = 16.6 |
| | 27 | 13 | 14 |

Of the fifteen cases in which the iridectomy was complete, three showed signs of iritis and twelve showed no signs of inflammation of the iris. On the other hand, of the twelve cases in which a partial or no iridectomy was performed, ten had iritis, while but two escaped. These results favor a complete iridectomy in the proportion of 20 to 83.3.

The results obtained by Colonel Elliot as tabulated in Table 5 justify the same conclusion.

Of the twenty cases in which an iridectomy had been made, either at the time of operation or previously, 10 per cent. showed signs of iritis; whereas in the fifty-six cases in which the trephining had been performed without or with partial iridectomy, iritis was present in 51.7 per cent. These results favor a complete iridectomy in the proportion of 10 to 51.7.

Of the six failures two (Cases 3 and 4) were cases of buphthalmos in the same patient. In each eye there was loss of vitreous at the time of operation, and it is possible that the opening was made too far back. Photophobia and keratitis, which were present at the time of operation, were very much improved, but ten weeks later there was in each eye a small white spot at the site of the trephine opening, and the tension had increased from 48 to 70 mm. in the right eye, and from 48 to 68 mm. in the left eye. The tension both before and after operation was taken under ether anesthesia. One case of glaucoma, secondary to cataract operation followed by uveitis (Case 14), was a very unfavorable case for any operative procedure. The tension at the time of the operation was 36 mm. Twelve days after operation it was 14 mm., but soon increased, until in two months' time it had reached 100 mm. At this time the eye was enucleated for relief of pain, the patient refusing to submit to any other operative procedure. The fourth case (Case 15) was probably one of hemorrhagic glaucoma. No ophthalmoscopic examination could be made, but the fellow eye showed a marked hemorrhagic retinitis. The last case (Case 16) was one of chronic inflammatory glaucoma, vision nil, deep scleral injection, very shallow anterior chamber, iris inflamed, tension 60 mm. A 1.5-mm. trephine opening was made associated with a small iridectomy, with no relief of tension. Later a 2-mm. opening was made with small iridectomy. The tension was temporarily reduced, but the eye was too tender to permit of tonometric observations. Two months after the second operation the tension was 60 mm., and there was a large prolapse of iris or ciliary body at the site of each trephine opening, one about 2 mm. and the other fully 3 mm. in diameter.

A comparison of the results obtained from an iridectomy and a sclerocorneal trephining operation is well shown in Case 11 (Chart 1), an advanced case of simple glaucoma in both eyes. Vision was reduced to light perception, tension right eye 75 mm., left eye 105 mm. Oct. 10, 1912, I performed a deep iridectomy on the right eye and trephined the left, using a 1.5-mm. trephine, and included the pupillary border of the iris in the iridectomy. Fifteen days later the tension in the right eye was 22 mm., in the left eye 18 mm. Five days later the tension in the right eye, which had been trephined, was 30 mm., but later it dropped to between 11 and 20 mm., where it has since remained. The eye on which an iridectomy was done gradually increased in hardness until on April 21, 1913, five months after operation, the tension was 60 mm. A sclerocorneal trephining operation was performed at this time and the tension reduced to 20 mm., where it remained for nine months, when, following a cataract extraction, it increased to 30 mm., but soon dropped to normal, where it has remained until the present time.

The results in a second case on which a comparative test was made and kept under observation for a period of two and one-half years is shown in Chart 2 (Case 10). The right eye, with a tension of 30 mm., was trephined on Oct. 10, 1912, and at the same time the left eye, with a tension of 35 mm., was subjected to a broad, deep iridectomy. Vision in each eye at the time of operation was light perception. The tension

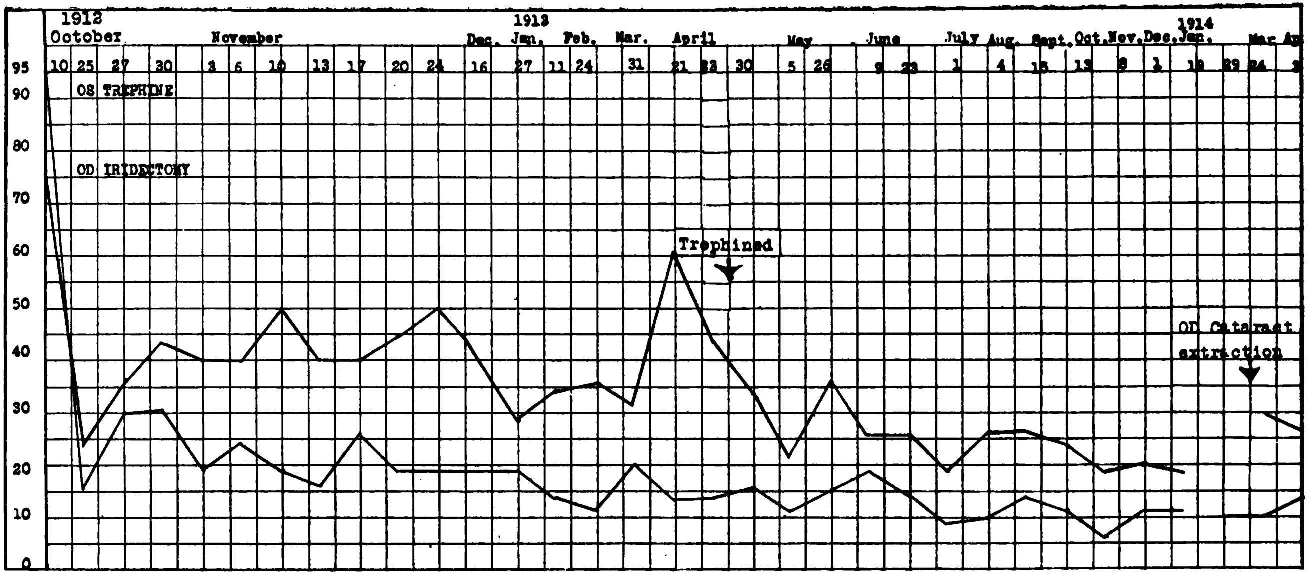

Chart 1.—Tonometric Records in Case 11.

of the eye which was trephined was reduced to between 4 and 10 mm., while that on which an iridectomy was done was reduced to between 20 and 30 mm. While the result in this case is recorded as good, it would be important to know what effect prolonged low tension would have on vision.

In Chart 3 is shown the tonometric result in a case of simple glaucoma in both eyes with an acute exacerbation in the left. An iridectomy in the left eye followed by a trephining operation failed to keep the tension down, and the eye was enucleated for the purpose of relieving pain. The tension in the right eye has remained normal since the time the iridectomy was performed.

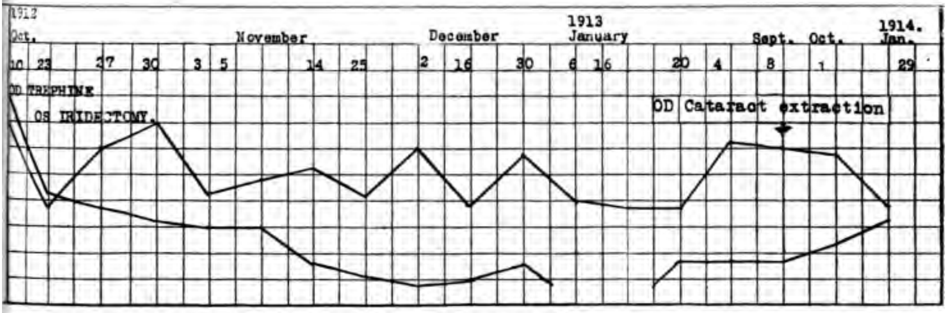

Chart 2.—Tonometric Records in Case 10.

## CONCLUSIONS

The average results obtained from the sclerocorneal trephining operation for relieving glaucoma are better in the non-inflammatory type than in the other forms of the disease.

The results obtained when the operation was associated with a complete coloboma were better, as regards both the frequency of the occurrence of iritis and the effects on the tension, than when performed with a partial iridectomy or when the iris was left intact.

The remote results may not be so good as those shown immediately after the operation.

The number of cases here recorded is not sufficiently large to permit of definite conclusions concerning the

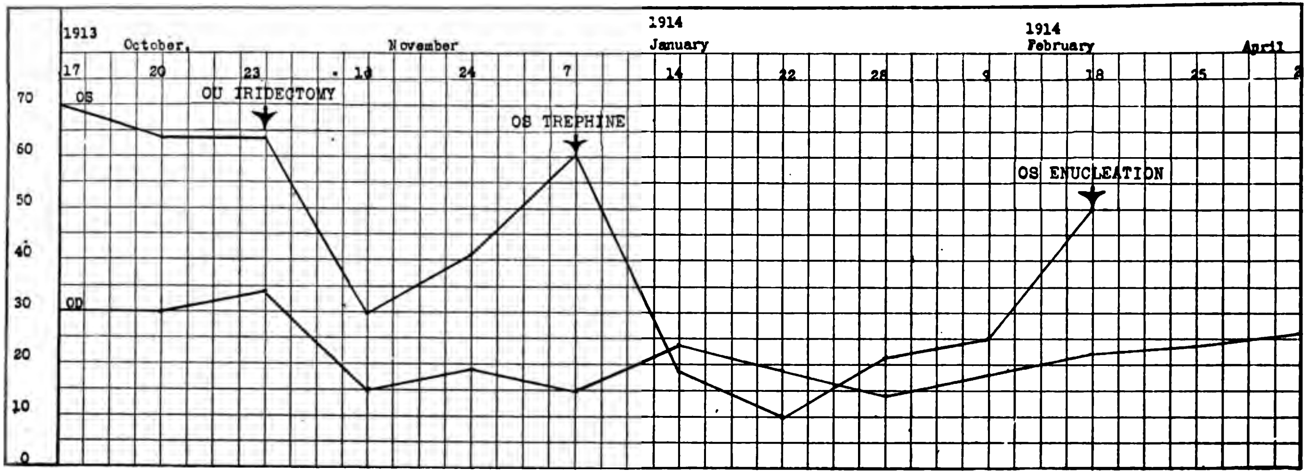

Chart 3.—Tonometric Records in Case 24.

real value of the sclerocorneal trephining operation in relieving glaucoma.

I am greatly indebted to Dr. D. V. Smith of the Ophthalmic Department of the University of Michigan for valuable aid in preparing the statistics presented in this paper.

32 Adams Avenue.

# MEMORANDA

# LATE INFECTION FOLLOWING A TREPHINING FOR GLAUCOMA *

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CHICAGO

Just a year ago Wagenmann reported the first case of "late infection" following a sclerectomy operation for glaucoma. Since that time twenty-five observers have added thirty-eight cases, and Herbert says that the growing number of late septic infections in Europe is causing much concern. It is therefore a little surprising that only one such observation[1] has been published in this country and, believing that all allied observations on any new procedure should be published until its disadvantages as well as its advantages are thoroughly understood, I present the following case:

*Patient.*—W. G. F., aged 56. Vision in the left eye began to fail two years ago, in the right eye three months ago. Two months ago the left eye was entirely blind. Colored rings were seen earlier before the left eye and are now seen before the right. Lues was acquired twelve years ago.

Jan. 13, 1913: The right cornea and media are clear, iris normal, pupil 4 mm. and reacts sluggishly; the disk shows only a physiologic cupping. The retinal veins are dark and slightly tortuous. Tension normal to touch and 20 mm. Hg by the Schiötz tonometer. Right vision 3/10—1, not improved by his +1.D glass. Form field fully normal. The left anterior corneal stroma and epithelium are clouded, although the iris shows through; the direct and consensual reactions of the 4 mm. pupil are gone, the fundus gives a red reflex but no details. Tension + 1; Schiötz = 50-55 mm. Hg. No perception of light.

Physostigmin (eserin) instilled into the eyes for a few days narrowed both pupils to less than 1 mm. The further effect

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[1] Gifford: Ophth. Rec., January 1914, xxiii, 1.

on the right eye greatly surprised me and proved that what I had considered to be a normal eye was in reality a glaucomatous one, for the tension decreased perceptibly to the touch and from 20 to 10 mm. according to the tonometer. The red field was now taken and found to be reduced to one-half, although the form field was normal, as before. The uncorrected vision improved from 3/10—1 to 6/10, and to 10/10 with his +1.D. The left cornea cleared and the disk was found to have a complete and total ampulliform excavation, all vessels disappearing beneath the overhanging scleral ring. Diagnosis: right and left simple glaucoma.

January 21: Trephine of the right scleral limbus with conjunctival flap and basal iridectomy.

February 7: Left subconjunctival injection of 4.5 per cent. sodium citrate to see if the tension would decrease.

February 8: Left tension not reduced.

February 11: Left tension not reduced (+ 3); cornea edematous. Physostigmin reordered.

February 12: Right tension normal to the fingers, cornea clear. The scleral limbus conjunctiva shows a 3 by 6 mm. flap-bleb straight up from the limbus between 10 and 12 o'clock. Vision, right + 1.5 = 8/10 — 4. Discharged with directions to continue the use of physostigmin in the left eye.

April 18: Patient returned twelve weeks after the operation, with the statement that three days before the right eye had suddenly become red and painful, the upper lid swollen, vision "foggy." The week before the patient had an acute cold.

Right eye: Photophobia, lacrimation and slight ciliary tenderness, marked ciliary and conjunctival injection, cornea clear. The site of the operation showed a yellowish, slightly elevated 4 mm. area between 10:30 and 12 o'clock. The anterior chamber was of normal depth, the 3 by 6 mm. coloboma above and the 4 mm. pupil area were filled with a cloudy exudate which obscured all view of fundus except for a small ½ mm. portion of the upper inner angle of the pupil, where a faint red reflex only could be seen. A hypopyon, 1 mm. high, 6 mm. broad, was present. The pupil did not respond to light or accommodation. The iris was dark green (left iris blue) and the vessels over the pupillary portion markedly injected. Tension 18½ mm.

Diagnosis: Suppurative iridocyclitis.

April 19: Chemosis bulbi. Atropin revealed a brown posterior synechia at 3:15 o'clock and considerable exudate on the lens.

Five days later, under added heat, dionin and potassium iodid, the chemosis and pain were lessened and the hypopyon had entirely disappeared; a thick pad of spongy exudate almost completely filled the pupil area, the cornea was slightly clouded, lid edema marked, tension not increased. Synechia going through all layers of the iris were found from 3 to 3:30 and from 8 to 9 o'clock.

May 10: Tension, increased for first time, not relieved by subconjunctive salt injection; anterior chamber everywhere abnormally deep; trephined area flattened. Vision, right 1/10. "Prognosis for vision bad."

May 17: Tension lower.

May 16: Vision, right 3/10; tension 40 mm. Left tension (physostigmin) 23 mm. Hg. Discharged.

## SUMMARY OF CASE

An eye affected with simple glaucoma was trephined and the patient made an uneventful recovery. Twelve weeks later the patient returned with the statement that he had caught cold the week before and that the eye had become red and painful four days later. The conjunctival flap-bleb, which measured 4 by 6 mm., was yellowish and a severe suppurative iridocyclitis was present. Under treatment the hypopyon promptly disappeared and the uveitis got well in seven weeks, but a moderate increase of tension (40 mm. Hg) supervened and vision decreased from 8/10—4 (three weeks after the operation) to 3/10. The conjunctival bleb flattened down to the scleral level.

The case agrees in all essentials with those reported as "late septic infection" following sclerectomy and is therefore not worthy of extended summary and discussion. For a review of the literature the reader is referred to the article by Gifford.[2]

It is to be regretted that fluorescin was not used to determine the presence or absence of a defect in the conjunctival flap, as was done by Axenfeld and by Harms.

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[2] This covers a wide range of experience in the hands of many competent observers and includes a discussion of the frequency of the infection, comparison between frequency after Lagrange's and after Elliot's operations, the interval, the influence of buttonholing the flap, the position of the trephine opening, the condition of the conjunctival bleb after operation and after infection, the factors of trauma, dacryocystitis and conjunctivitis, the use of fluorescin to detect a break in the flap, the relation of the pus beneath the flap to that within the eye, the resulting suppurative iridocyclitis and the course of the same, rises in tension, panophthalmitis, phthisis bulbi, sympathetic ophthalmia, histologic examination of the wound tract (Schur's case), Kuhnt's transplantation after buttonholing, cautery of flap, application of tincture of opium to toughen the flap, use of Roemer's antipneumococcus serum, etc.

The reports of cases published since the appearance of Gifford's article are those of Cramer, Herzau, Stock (second report), Bachstez, Herbert, Constantinesco and Pooley.

## BIBLIOGRAPHY

Wagenmann: Ber. d. ophth. Gesellsch. zu Heidelberg, 1813, p. 211.

Isakowitz: Klin. Monatsbl. f. Augenh., 51, N. F., June, 1913, xv, 814.

Axenfeld: Klin. Monatsbl. f. Augenh., 51, N. F., June, 1913, xv, 816.

Schur: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 377.

Harms: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 380.

Pagenstecher: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 388.

Stock: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 402.

Story: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 403.

Bronner: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 404.

Wicherkiewitz: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 388.

Morax: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 388.

Elschnig: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 595.

Stock: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 772 (second report).

Cramer: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 771.

Paul: Klin. Monatsbl. f. Augenh., 51, N. F., 1913, xvi, 771.

Kuhnt: Ztschr. f. Augenh., 1913, xxx, 137.

Meller: Klin. Monatsbl. f. Augenh., January-June, January, 1914, p. 1.

Bachstez: Klin. Monatsbl. f. Augenh., January-June, January, 1914, p. 112.

Pooley: Wchnschr. f. Therap. u. Hyg. d. Auges, Aug. 7, 1913, p. 346.

Constantinesco: Clin. Ophthalmologique, 1913, p. 689.

Fehr: Quoted by Gifford as in Wchnschr. f. Hyg. u. Therap. d. Auges, p. 85, Nov. 13, 1913 (error?).

Gifford: Ophth. Rec., 1914, xxiii 1.

Herbert: Ophth. Rec., 1914, xii, 2.

Herzau: Klin. Monatsbl. f. Augenh., N. F., 1913, xvi, 773.

# MEMORANDA

# MEMORANDA

# THE VALUE OF MIOTICS IN CHRONIC GLAUCOMA *

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I have already treated of this subject before the Section on two previous occasions, at the Boston session in 1906 and the Chicago session in 1908, and this third paper is largely supplementary to these earlier communications.

At the 1908 meeting, sixty-five cases of simple chronic glaucoma were analyzed which had been treated by miotics, of which all but seven had been observed over a period of two years, and twelve for more than ten years, and the results attained by the constant and prolonged use of these drugs were compared with a similar series of cases treated by iridectomy. Among the percentages deduced from this analytic study it was found that central vision was maintained in the miotic series, each case of which was observed over an average period of five years and eight months, in 80 per cent. of the cases, in comparison with 25 per cent. in the most favorable group of iridectomized eyes. Other figures bearing on other phases of the glaucoma problem were equally conclusive as to the superiority of the drug treatment and demonstrated conclusively that miotics exert a much more beneficial influence on the progress of simple chronic glaucoma than the removal of a portion of the iris. Whether or not better results will be found to follow the newer operations for glaucoma, time and the careful analysis of a large series of cases treated by these more recent methods can alone determine; and though it would appear that the establishment of a permanent filtration scar by the trephine gives

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greater hope of retaining vision in a higher percentage of cases than other operative means, it must be remembered that the claims for this procedure have not yet been proved, and some years must still elapse before a true estimate may be had of the value of this now popular measure.

Of the sixty-five cases analyzed by me in my 1908 series, twenty-two were taken from my own records, the remainder contributed by generous colleagues. During the six years which have elapsed since the publication of that report, nine of my twenty-two patients have either died or passed from under my observation; thirteen, however, still remain under my care, and their further progress, with the histories and analyses of eighteen additional cases, also from my private practice and all observed over a prolonged series of years, will form the basis of the present communication.

Of the 13 cases remaining from the 1908 series, 1 was observed for a period of 21 years, 3 for 18 years, 1 for 16 years, 2 for 14 years, 2 for 12 years, 1 for 10 years, 1 for 8 years, 1 for 7 years and 1 for 6½ years. In this series but 22 eyes lend themselves to analysis, as 3 were blind when they came under observation and one had been iridectomized.

Of the 22, 17 eyes were either normal or showed but the earliest traces of glaucoma at the first examination, in 3 the disease was moderately advanced, while in 2 the glaucomatous process had progressed to such an extent that but little vision remained. Fourteen eyes of this first group retained practically normal vision and field as long as they were under observation, a general average of ten years, as follows:

One eye presented practically normal vision and field after 21 years, 5 eyes after 18 years, 1 after 16 years, 1 after 14 years, 2 after 12 years, 1 after 10 years, 2 after 8 years and 1 after 7 years.

Three went blind while under treatment, in 5, 6 and 10 years, respectively. Of the 3 eyes in which the disease was but moderately advanced, 1 went blind in 2 years; the others have maintained vision 8 and 10 years, respectively. The results attained in the 2 eyes in which the disease was farthest advanced were strikingly good, vision and field being maintained without further loss in 1 case for 10 years, while in the second but a slight loss was recorded during the same period.

Of the 18 cases now reported for the first time, 1 was observed for a period of 18 years, 2 of 10 years, 1 of 8 years, 1 of 7 years, 7 of 6 years, 1 of 5 years, 2 of 3 years, 2 of 2 years and 1 of 1 year.

These 18 cases presented but 24 eyes for analysis, some of the patients having already lost one from glaucoma before consultation, while in others but 1 eye was affected. Of these 24 eyes, 13 showed but the rudiments of the disease at the first examination, the glaucomatous process was moderately advanced in 8, while in 3 central vision and the visual field were greatly compromised. In the first group practically normal vision has been maintained while the cases have been under observation, for periods ranging from 5 to 18 years, an average of 7½ years. In the second group of 8 eyes, vision has been maintained for periods ranging from 2 to 10 years, an average of about 4 years, while in the 3 eyes which were in the most advanced stage of the disease, vision has been maintained for periods ranging from 1 to 6 years, an average of 3 years. It should be noted that in 2 out of these 3 eyes the disease was of a pronounced hemorrhagic type and that notwithstanding this, vision is still maintained in 1 after a period of 6 years and that blindness ensued in the other after the maintenance of 6 years of fair vision.

The figures obtained by the analysis of these 31 cases agree in large measure with those published in connection with the 1908 series. As it was pointed out at that time, just as the advocates of operative measures find beginning cases to be more amenable to treatment than those in the more advanced stages of the disease, so cases treated by miotics from the early stages give better percentages so far as the maintenance of central and peripheral vision is concerned. Twenty-seven of the 46 eyes analyzed in the two groups of cases belonged to this class, and with the exception of 3, all preserved nearly normal vision and fields as long as they remained under observation, and this is the more extraordinary when it is considered that 1 of these cases has been observed for 21 years, 4 for 18 years and 8 for a period of 10 years or more. As I remarked in a previous paper, in view of such favorable statistics from the use of miotics, when the risks and complications attending all operations on the eye are considered, as well as the possibility of error in the diagnosis and operation being performed on eyes with merely atrophic nerves, is operation ever justified when both central and peripheral vision are normal, without trial of what miotics can do?

In 11 eyes in which the disease was noted as being moderately advanced, in the first group, 2 maintained vision for 8 and 10 years, respectively, and 1 went blind in 2 years, in the second group vision has been maintained for periods ranging from 2 to 10 years, an average of about 4 years. The best results of all, relatively speaking, were attained in the far-advanced or desperate cases, for of the 5 cases so designated, vision and field were maintained without further loss in 1 case for 10 years, and in a second with but slight loss during the same period. In the remaining 3 eyes, vision has been maintained for periods ranging from 1 to 6 years, and this in spite of the fact that in 2 of the latter the disease had assumed a hemorrhagic type. I would recall, in this connection, the remarkable results attained in Case 48 of the 1908 series, in which full central vision was maintained in a typically glaucomatous eye with field cut almost to fixation for 14 years.

It is in this class of cases that iridectomy has been attended with such bad results, and while cyclodialysis and the trephining operations may be less injurious, I shall continue to employ miotics continuously and energetically until convinced by the report of a series of suitable cases that operative measures have proved their superiority. My observations have convinced me that miotics cannot be regarded in any sense as curative, for notwithstanding their continuous use, the glaucomatous process still goes on, very slowly, it is true, but the eye grows steadily harder, the excavation becomes broader and deeper and the anterior chamber shallower. Again, I desire to emphasize what I have already said elsewhere, namely, that miotics should be relied on as the sole means of treatment only in those cases which are free from attacks of so-called glaucomatous congestion, the presence of such congestive symptoms being in my opinion the chief indication for some form of operative treatment, be it iridectomy, cyclodialysis or trephining; and second, that to gain the full benefit of miotics it is necessary that they should be administered properly. Beginning in doses small enough to avoid creating spasm of the ciliary muscle, and rapidly increasing the dose until the pupil of the affected eye is strongly contracted, this degree of contraction should be maintained as long as life lasts by gradually increasing the strength of the solution, from time to time, and by instillations of the drug at intervals of every three or four hours.

The miotics which are best adapted to control intraocular tension are physostigmin (eserin) salicylate and pilocarpin nitrate. This salt of physostigmin is more persistent in its effects and less changeable in solution than other salts of the drug and is less irritating to the conjunctiva. I prescribe a solution of pilocarpin to be used about every four hours, morning, noon and evening, and one of physostigmin of twice the strength at bedtime, thereby avoiding in a measure the blurring of vision which is occasioned by the action of the physostigmin on the ciliary muscle during the day, while the eye receives the greatest effect of the drug during the eight hours or more which elapse between the instillations of the drops during the night. In incipient cases of the disease an excellent initial dose is that of ⅙ grain of pilocarpin to the ounce of water. The strength should be gradually increased, so that at the end of a year 1 grain to the ounce is employed, at the end of the second year 2 grains, and at the end of the third year 3 grains to the ounce solution. This strength will suffice to maintain the pupils at the desired point of almost pin-point contraction. Physostigmin should be employed in half the strength of pilocarpin and should be increased in solutions of equal proportions.

Conjunctival irritation can usually be avoided by employing only fresh and sterile solutions of the miotics and by frequent cleansing of the conjunctiva by boracic acid solution. Should such irritation arise, local applications of argyrol and flushing of the conjunctiva with mild lotions, conjoined with the use of ice compresses and a weakened dose of the miotic will usually occasion its prompt disappearance.

Gentle massage of the eyeball is of decided advantage and should be practiced several times each day, for five minutes at a time. In addition to these local measures, the patient should be instructed as to the number of hours daily the eyes should be used in near vision. All near work should, of course, be restricted, and should be carried on only under the most favorable conditions regarding illumination, posture of the patient, etc.

Proper lenses should be adjusted to the eyes for both near and far use, and the refraction should be frequently estimated and any error corrected, changes in the refraction being rendered frequent by the action of the miotic on the ciliary muscle.

A large number of hours daily should be spent in the open air, and, as the disease is frequently the ocular expression of chronic rheumatism and gout, the patient should be urged to spend the greater part of the year under the most favorable climatic conditions to combat that diathesis. The skin should be kept active, the gastro-intestinal apparatus regulated, and any local source of inflammation or irritation, neighboring on the eyes, such as inflammation in the nasal passages or their accessory sinuses, should be allayed. Particular care should be given to the peripheral circulation, nitroglycerin and strophanthus being often of value. Strychnin should be administered, not only on account of its action on the circulation, but also because of its influence on the optic nerve. On account of their antirheumatic properties, the salicylates are of decided value and should be administered frequently for continued periods.

The notes of the thirty-two cases follow. These are necessarily much abbreviated and give but the briefest description of the progress of each case. The fields are reproduced in outline merely, and though in many instances a careful test of the visual field was made every three or four months, on account of the necessity for brevity, but one set of fields is published for each year or series of years. The outlines were obtained by testing with 10 mm. white and red objects, the outer representing the white field, the inner the red. All reference to the dosage of the miotics has also been omitted, the treatment prescribed in each instance having been along the lines specified in the preceding paragraphs.

## REPORT OF CASES

CASE 1 (Case 1 of 1908 series).—Woman, aged[1] 81, under observation nearly twenty-one years, first sought treatment on account of secondary glaucoma in the right eye, following iritis. Iridectomy was performed, but without avail, vision soon being reduced to 2/40 eccentrically, and the field being much contracted.

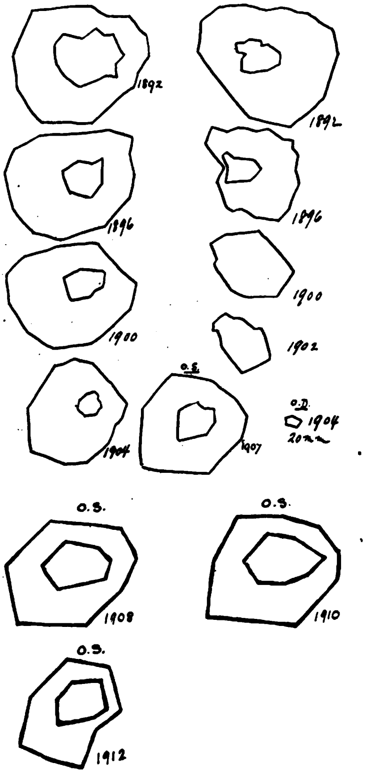

Fig. 1.—Fields of Case 1.

*Examination.*—O. S., signs of chronic glaucoma, anterior chamber shallow, sclera rigid, commencing pathologic excavation.

O. S. V. + S. 0.50 D. ⊃ + C. 0.25 D. ax. 20° = 5/5.

Field normal. This patient has been seen at intervals of at least every six months during twenty-one years, the miotics being gradually increased in strength until pilocarpin, 4 grains to 1 fluidounce, 2 drops three times a day, is now employed to keep the pupil contracted ad maximum.

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[1] The age given in all of the cases refers to what it was at the time of last observation.

During the past six years the disease has progressed but little. Vision equals 5/6, field but moderately cut. Reduction in vision and field doubtless are due to lenticular haze. This patient has been most assiduous in carrying out the treatment, and the splendid results must be attributed to that. At no time in the twenty-one years has there been any conjunctival irritation from the drugs.

*Remarks.*—Glaucomatous process held in check while under observation for almost 21 years, central vision being still normal and the field but slightly contracted.

Case 2 (Case 5 of 1908 series).—Woman, aged 64, under observation eighteen years. Full vision still maintained. Field but slightly contracted. Some tendency to conjunctival irritation, readily combated with astringents. At first examination in 1896:

O. D. + S. 1.75 D. ⌣ + C. 0.75 D. ax. 100° = 5/5.
O. S. + S. 1.25 D. ⌣ + C. 0.50 D. ax. 60° = 5/5.

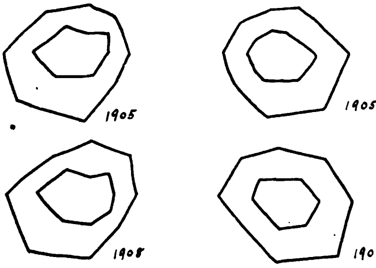

Fig. 2.—Fields of Case 2.

Both optic nerves hyperemic, with central excavations. Patient seen at intervals of one or two years for change of glasses and minor symptoms. Eight years after first observation, both optic nerves were noted as being pale and "luminous" and the excavations were deep and undermined above, this being the more pronounced in the right eye. The anterior chambers were shallow and the sclera rigid. Vision and fields normal. The strength of the miotics has been gradually increased, the pupils being maintained at pin-point contraction, but the excavations have gradually extended until they are now pronouncedly glaucomatous. Vision and fields practically normal in each eye.

*Remarks.*—Development of glaucoma cups while under observation. Stage of hyperemia of the nerve preceding the atrophic process. Maintenance of full visual acuity and extent of visual fields with miotics.

Case 3 (Case 20 of 1908 series).—Woman, aged 86, under observation eighteen years. At time of first observation complained of occipitofrontal pains radiating into the eyes, worse on the left side. Six months previously she had had several attacks of sudden failure of vision in the left eye, which had lasted for a few minutes. During these periods of visual obscuration, the defect began in the temporal field. Both nerves were hyperemic, central excavation in the right eye becoming undermined down and out. Excavation in the left eye shallow but typically glaucomatous. Anterior chambers shallow. Sclerae rigid, especially in O.S.

At the end of two years, vision had deteriorated in O.S. to 3/60, and at the end of another year to 1/60, at which it still remains.

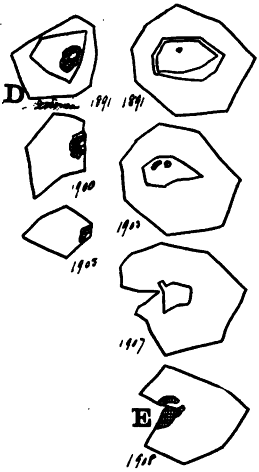

Fig. 3.—Fields of Case 3.

1901: O. D. V. + C. 0.25 D. ax. 70° = 5/5.
1902: O. D. V. — S. 0.25 D. ⊃ — C. 0.50 D. ax. 120° = 5/5.
1903: O. D. V. — S. 0.50 D. ⊃ — C. 0.75 D. ax. 145° = 5/5.
1905: O. D. V. — S. 1. D. ⊃ — C. 1.25 D. ax. 145° = 5/5.
1906: O. D. V. — S. 2. D. ⊃ — C. 1.25 D. ax. 145° = 5/5.
1907: O. D. V. — S. 1.25 D. ⊃ — C. 1.25 D. ax. 145° = 5/5.

This patient was seen once in 1908 and again in 1911, though the miotics were employed continuously in the interim. In 1908, vision equaled 5/9 in the remaining eye (O. D.), and in 1911, 5/15. The fields were not taken on either of these occasions. As the patient was 94 years of age when the last examination was made and as there was considerable haze of the lens, the result must be regarded as being most satisfactory.

*Remarks.*—Glaucoma held in abeyance in O. D. for 18 years, in O. S. five or six years, by miotics. It is probable that a more favorable result would have been gained in O. S. had an interval of several years not elapsed during which the miotics were used irregularly. At times, during the course of the disease, subacute symptoms arose, which were promptly made to disappear by stronger doses of pilocarpin.

CASE 4 (Case 4 of 1908 series).—Man, aged 51. Under observation 18 years. At time of first observation, both optic nerves hyperemic, especially the left.

O. D. + S. 0.50 D. ⊃ + C. 0.75 D. ax. 90° = 5/5.
O. S. + S. 0.50 D. ⊃ + C. 1. D. ax. 70° = 5/5.

Fields of vision normal. Left-sided neuralgia at times, with some pain in corresponding eye. Seen at intervals of one or two years. Eight years after first observation excavations in each eye noted as being suspicious, and physostigmin, $\frac{1}{12}$ grain to the ounce, 2 drops each day, prescribed. Two years later excavations more pronounced, especially in O. D. Vision and field of vision normal in each eye. At present quite a deep glaucomatous excavation in O. D., extending to scleral ring externally. The excavation in O. S. has also become glaucomatous, being more extended but not so deep as in the fellow eye. Some contraction of O. S. field to nasal side. Tonometer shows O. D. 35 mm. Hg, O. S. 25 mm. Hg.

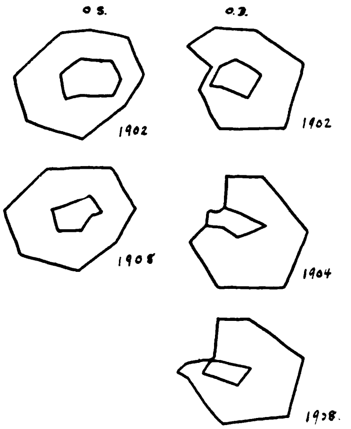

Fig. 4.—Fields of Case 4.

CASE 5 (Case 6 of 1908 series).—Woman, aged 79, under observation sixteen years. Gradually failing vision in each eye for some months at time of first consultation. Gouty. Suffers from nasal catarrh. Both optic nerves hyperemic, with large temporal excavations. Sclera rigid. Anterior chambers shallow. Fields of vision normal.

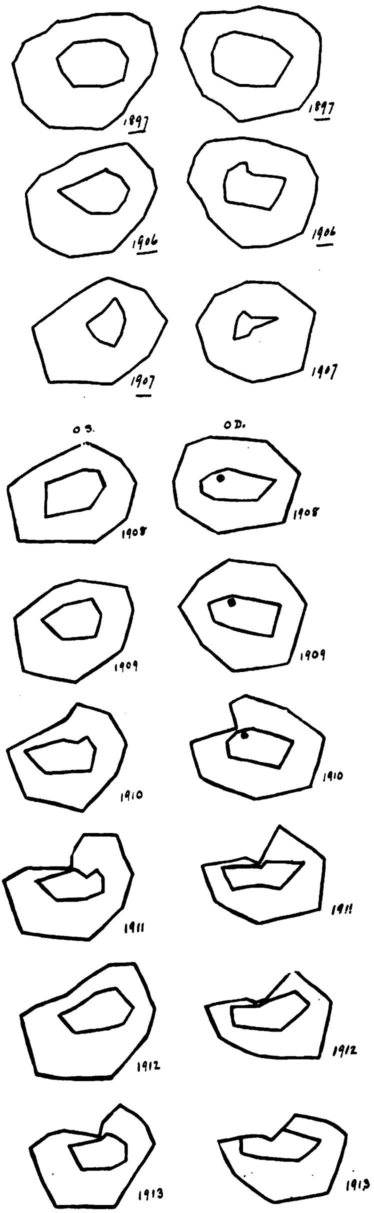

Fig. 5.—Fields of Case 5.

O. D. V. + S. 1.75 D. ⊃ + C. 0.50 D. ax. 30° = 5/5.
O. S. V. + S. 1.25 D. ⊃ + C. 0.75 D. ax. 60° = 5/5.

The patient has been seen at intervals of six months, and, though the nerves have become somewhat atrophic and large shallow glaucomatous cups have developed in each eye, the anterior chambers shallower and tension increased, central vision and the fields of vision have remained almost normal. Considerable haze of O. D. lens with some involvement of pupil area.

*Remarks.*—Glaucoma developed under observation, despite use of miotics, but vision and visual field have remained normal.

Case 6 (Case 9 in 1908 series).—Woman, aged 74, under observation fourteen years. Gouty. Has always suffered from head pain. At time of first consultation nerves gray; shallow physiologic excavations, that in the left eye with undermined edges. Sclera rigid. Fields normal.

O. D. + S. 0.75 D. ⊃ + C. 0.50 D. ax. 50° = 5/5.
O. S. + S. 0.75 D. ⊃ + C. 0.25 D. ax. 120° = 5/5.

Second observation, a year later, when excavation in O. S. was found to be deeper and more glaucomatous in type. Head pain had persisted despite use of glasses, but there was no obscuration of vision in either eye. Vision and field still normal.

The patient then passed from observation for four years, although she had been warned of the necessity of frequent observations of the left eye and told of its glaucomatous nature. At the visit, four years later, complaint of failing vision in O. S. for a year or more was made and also of a continuance of the head pain. Examination showed a deep typical glaucomatous cup in the left eye and a shallow glaucomatous one in the right. Both nerves quite gray and walls of retinal vessels thickened. Pilocarpin, 1 grain to 1 fluidounce three times a day, with strychnin and nitroglycerin.

O. D. V. + S. 1.5 D. ⊃ + C. 0.50 D. ax. 30° = 5/5.
O. S. V. — C. 0.50 D. ax. 30° = 5/7½.

During the past six years, vision and field of vision have steadily deteriorated in each eye, that in the right eye now equaling 5/15 (— S. 0.50 D. ⊃ — C. 0.62 D. ax. 110°), that of the left hand-movements in the temporal field only. There is a moderately deep cup in the right eye and tension is decidedly elevated.

*Remarks.*—This case illustrates that the glaucomatous process may arise in an eye in which miotics are being continuously employed, and progress, though slowly, to a considerable degree. By reason of advanced intra-ocular changes, this case was thought to be inoperable, an opinion in which Mr. E. Treacher Collins of London, in consultation, concurred, so that while the results obtained from the miotics are not all that could be desired, nevertheless they have stayed the progress of the disease very markedly, and useful vision is still maintained in one eye.

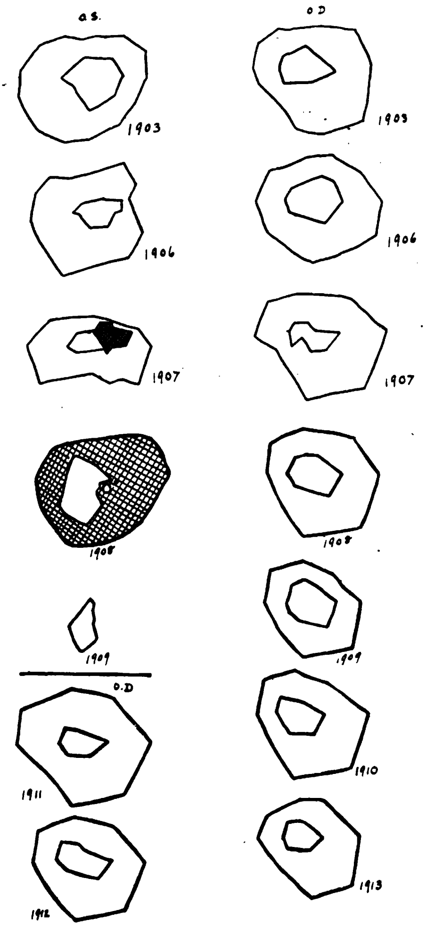

Fig. 6.—Fields of Case 6.

Case 7 (Case 30 of 1908 series).—Woman, aged 83, under observation fourteen years. History of failing sight for two years. Rheumatic. Large glaucomatous excavations in each eye, more pronounced in the left eye. T. + in each eye.

O. D. V. + S. 0.50 D. = 5/5 partly.
O. S. V. + S. 0.50 D. ⊂⊃ + C. 0.50 D. ax. 145° = 5/5 partly.

*Remarks.*—As the years have passed, tension has steadily risen, with all the attendant signs of a steadily progressive chronic glaucoma. Even at the first examination, sclerosis of the retinal vessels was marked and the circulation of the optic nerves manifestly poor. Two years ago, the left eye became suddenly blind from thrombosis of the central artery of the retina, and within the past few months the temporal half of the right field has been lost as part of a hemiplegia resulting from a cerebral thrombosis. Throughout the case, there has been the complication of a large mucocele of the frontal cells, which has fortunately at all times been kept freely drained by nasal treatment. Miotics have been persistently administered, without causing any conjunctival irritation.

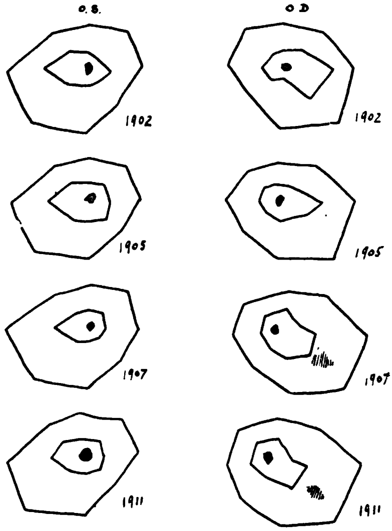

Fig. 7.—Fields of Case 7.

CASE 8 (Case 10 of 1908 series).—Woman, aged 77, under observation twelve years. At time of first consultation, vision said to have failed in each eye during the six months previous. Both nerves hyperemic, with large excavations becoming undermined to nasal side. Venous pulse. Anterior chambers shallow. Sclera rigid. Fields normal.

O. D. V. + S. 2.5 D. ◯ + C. 0.25 D. ax. 145° = 5/5.
O. S. V. + S. 2.5 D. ◯ + C. 0.25 D. ax. 160° = 5/5.

At end of two years cups had assumed a typical glaucomatous appearance, and tension was appreciably elevated in each eye. Central vision still normal, but form fields somewhat compromised concentrically, that for red being decidedly restricted.

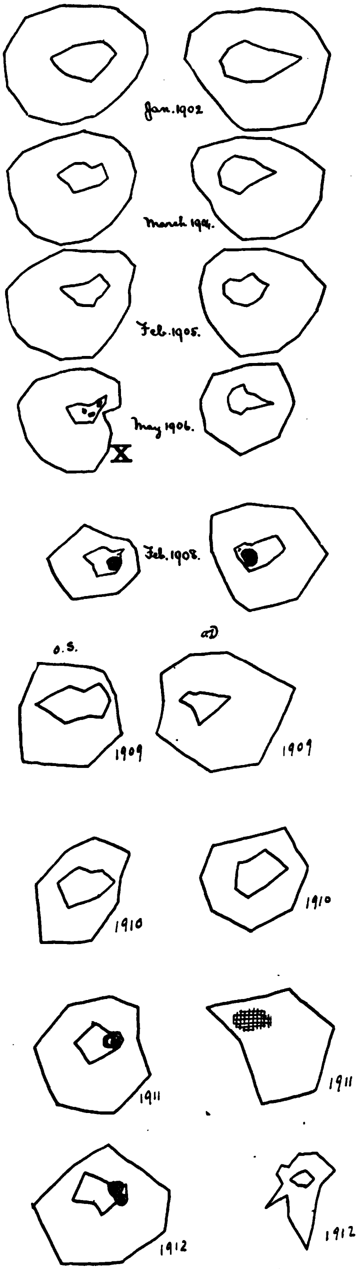

Fig. 8.—Fields of Case 8.

At end of six years, moderately deep glaucoma cups, anterior chambers quite shallow. Tension elevated.

O. D. V. + S. 0.25 D. ⌢⌣ + C. 0.62 D. ax. 30° = 5/5.
O. S. V. + S. 1.25 D. ⌢⌣ + C. 0.37 D. ax. 30° = 5/5.

During the past six years, there has been a steady progression of the disease with an increasing lenticular haze. Vision reduced to 5/35 in O. D. and to 5/15 in O. S. This loss is not altogether attributable to the glaucoma, however, as the commencing cataracts undoubtedly obscure vision.

*Remarks.*—The patient, now aged 77, has been under observation since she was 65 years of age and still has useful vision. If central vision deteriorates further, iridectomies will be advised, with a view to enlarging the pupils, possibly decreasing tension, and to pave the way for the delivery of the cataractous lenses.

CASE 9 (Case 17 of 1908 series).—Woman, aged 61, under observation twelve years. At time of first observation, dimness in left eye of six weeks' standing. Gouty. Mother had glaucoma. Both optic nerves gray; central excavation in the right eye and a forming glaucomatous one in the temporal half of the left nerve; sclera rigid, especially in O. S.; both anterior chambers shallow.

O. D. V. + S. 0.50 D. ⌢⌣ + C. 0.37 D. ax. 110° = 5/5.
O. S. V. + S. 0.50 D. ⌢⌣ + C. 0.37 D. ax. 70° = 5/5.

During the past six years there has been but little progress in the disease. Vision is still normal in each eye and the field is but little more restricted than it was seven years ago. Miotics have been used constantly always with a tendency to create conjunctival irritation. Patient lives at a distance, and is doubtless not careful to use only fresh and sterile drugs.

*Remarks.*—Glaucoma controlled in left eye, despite the considerable involvement of nerve which was present when the patient first came under observation. Right eye still normal.

CASE 10 (Case 32 of 1908 series).—Man, aged 70, under observation ten years. Poor vision in right eye for some years past, following dynamite explosion. Vision of left eye totally lost at that time. O. D. V. worse past six months. Halos at times. Large glaucomatous excavation in right eye; anterior chamber shallow; pupil large; cornea maculated in pupillary area; T. +. Left eye, phthisis bulbi.

O. D. — S. 1. D. ⌢⌣ — C. 1.5 D. ax. 180° = 5/9.

Seen at intervals of six months, miotics being increased from time to time. Now using pilocarpin, 3 grains to 1 fluidounce, 2 drops at night. During the ten years of treatment vision has practically held its own, that is, 5/15. There has been considerable loss in the nasal field, but the temporal field remains full.

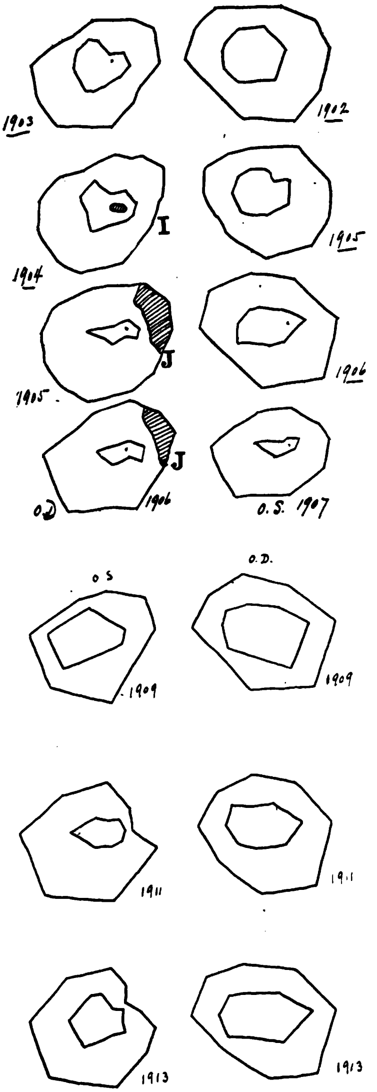

Fig. 9.—Fields of Case 9.

*Remarks.*—This result is exceptionally good, for the patient lives at a distance and cannot give so much care as is desirable to the conjunctiva. His health, too, has been bad, his vitality having been greatly lowered by repeated attacks of dysentery.

Case 11 (Case 41 of 1908 series).—Man, aged 57, under observation eight years. At time of first consultation vision in right eye had been misty six months. Halos at times. Rheumatic history. Had been under ophthalmic care, but no miotics had been employed. There was a large glaucomatous excavation in right eye, with a shallower but also characteristic excavation in the left. Tension markedly elevated in both eyes. Anterior chambers shallow.

O. D. V. + C. 1. D. ax. 20° = 5/15.
O. S. V. + S. 0.25 D. ⊃ + C. 0.50 D. ax. 180° = 5/5.

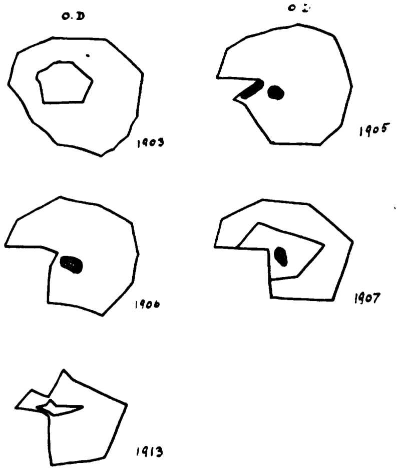

Fig. 10.—Fields of Case 10.

Despite continuous use of miotics, vision gradually failed in O. D.; in 1909 to 5/35, in 1910 to 5/100, the reduction being occasioned by slight subacute exacerbations of the glaucoma. Operative interference rejected on account of hemorrhagic type of the disease. For the past two years, the right eye has been practically blind. Vision in the left eye has been maintained at normal and the field of vision is normal. Tension, however, has increased and the cup has deepened.

*Remarks.*—Results from miotics disappointing in right eye, good in the left. Perhaps iridectomy in the right eye would have conserved vision longer.

Case 12 (Case 19 of 1908 series).—Woman, aged 69, under observation seven years. At time of first consultation, right

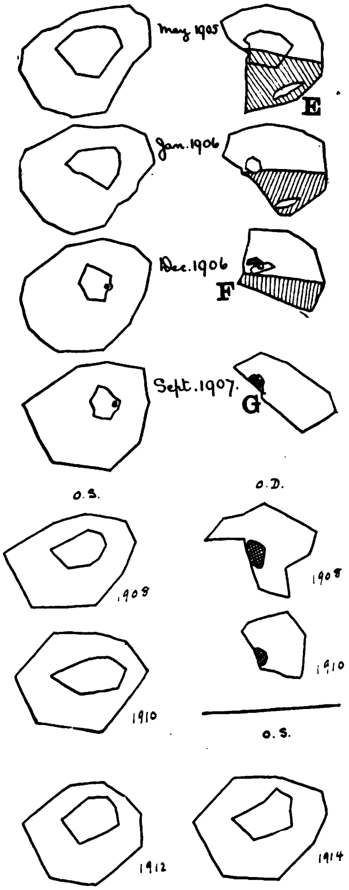

Fig. 11.—Fields of Case 11.

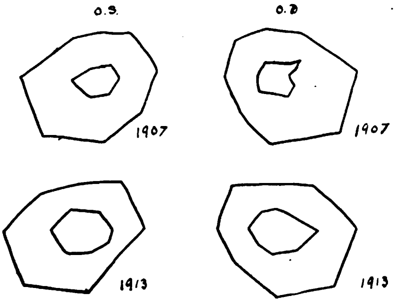

Fig. 12.—Fields of Case 12.

eye just recovering from an iridectomy which had been done by another surgeon on account of acute glaucoma. Shallow excavation in head of optic nerve, but eye quiet and no glaucomatous symptoms. Left eye free from glaucoma, save for a doubtful excavation to outer side of nerve. Anterior chamber in each eye shallow. Tn. Fields of vision normal in each eye. Gouty subject.

O. D. V. + S. 1.5 D. ⊃ + C. 1.75 D. ax. 160° = 5/5.
O. S. V. + S. 2.5 D. ⊃ + C. 0.62 D. ax. 90° = 5/5.

Glaucoma has made but little progress in either eye, as is evidenced by normal vision and fields. Miotic has been used in the operated eye as well as in the fellow, so that the maintenance of good vision in that eye may not be wholly the result of the iridectomy.

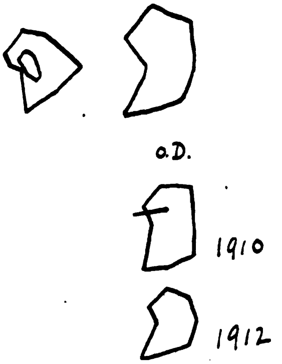

Fig. 13.—Fields of Case 13.

*Remarks.*—Despite glaucomatous outbreak in right eye, no glaucomatous symptoms have appeared in either eye, though a pathologic excavation has formed in the left eye, even though the pupil has been maintained at pin-point contraction.

Case 13 (Case 54 of 1908 series). Woman, aged 77, under observation six and one-half years. Poor health. Advanced vascular degeneration, vision in the left eye failing five years, in the right three years. Had never used miotics, though under the care of an eye specialist for five years. At time of first consultation:

O. D. + S. 0.50 D. ⊃ + C. 0.50 D. ax. 180° = 5/7½.
O. S. V. faint light perception only.

Observation of this patient continued over a period of five years following the last report, and notwithstanding her advanced age (77 years at time of last observation in 1912), very high intra-ocular tension, the conjunctival irritation excited by the miotics, and insufficient home care, vision in O. D. has been maintained to a most remarkable degree, namely:

1907: O. D. + S. 0.50 D. ⊃ + C. 0.50 D. ax. 180° = 5/6.
1908: O. D. — S. 0.50 D. ⊃ — C. 0.50 D. ax. 90° = 5/9.
1909: O. D. — S. 1. D. = 5/12.
1910: O. D. — S. 1. D. = 5/12.
1911: O. D. — S. 1.5 D. = 5/35.
1912: O. D. — S. 1.5 D. = 5/40.

As shown by the accompanying sketch, the field of vision in the right eye was much reduced even at time of first examination.

*Remarks.*—This case represents the power of miotics to retain vision for a long period even under the most desperate conditions, vision being maintained for six and one-half years in a person of very advanced years and great debility, the field of vision being greatly contracted and vision rapidly deteriorating.

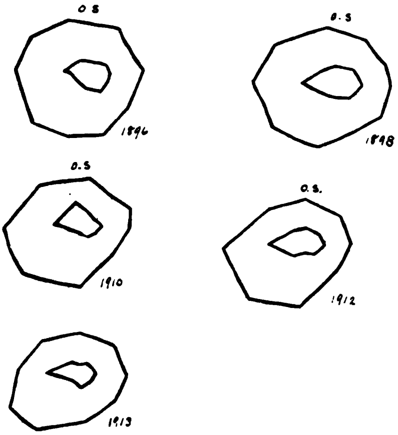

Fig. 14.—Fields of Case 14.

Case 14.—Woman, aged 67. First consultation June 3, 1896.

O. D. V. reduced to 1/50.
O. S. V. — S. 1. D. ⊃ — C. 3. D. ax. 10° = 5/5.

Subacute glaucoma in O. D., deep cup. Commencing excavation in left eye. Iridectomy O. D., resultant vision of 2/40. O. D. totally blind two years after operation. O. S. V. now 5/7½ with — S. 0.75. D. ⊃ — C. 4. D. ax. 15°. Field somewhat concentrically contracted. Moderately deep cup. Tonometer = 38 mm. Hg. Pilocarpin has been used almost uninterruptedly during the past eighteen years, and despite the advanced age of the patient, vision is still almost normal and the field of vision fair.

*Remarks.*—In view of the loss of the right eye from inflammatory glaucoma, the control of the disease by miotics seems the more remarkable.

CASE 15.—Woman, aged 64. First consultation November, 1904. Poor vision in O. S. for five years. Examination showed O. D. to be normal, O. S., seat of subacute glaucoma of a hemorrhagic type.

O. D. V. = 5/5
O. S. V. = 5/35

Iridectomy O. S., with resultant vision of 2/60.

During the past five years the left eye has remained *in statu quo*, while the glaucomatous process has progressed though ever so slightly in the right. Vision is still normal and the field of vision normal. During the last two years, there has been more or less irritation from the miotics, this condition being favored by a certain amount of obstruction in the lacrimal passages.

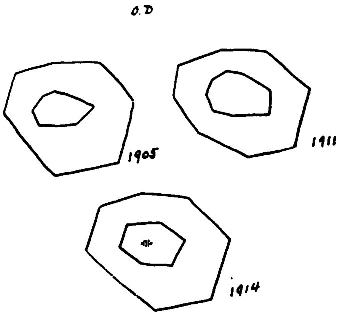

Fig. 15.—Fields of Case 15.

*Remarks.*—Notwithstanding the tendency to a hemorrhagic type of glaucoma, in the right eye, it remains practically unaffected with normal vision.

CASE 16.—Man, aged 56. First consultation ten years ago. Failing vision in O. S. past year.

O. D. + S. 0.75 D. = 5/5.
O. S. + S. 1.25 D. ⊃ + C. 0.25 D. ax. 160° = 5/5.

Commencing glaucoma cup in O. D., pronounced one in O. S. T. + 1 in O. S. Considerable sclerosis of retinal vessels. Miotics prescribed in suitable dose. Patient has been seen at irregular intervals during the last ten years, but miotics have been used with great perseverance and faithfulness. Central and peripheral vision is still normal. Both cups now unmistakably glaucomatous, that in the left eye

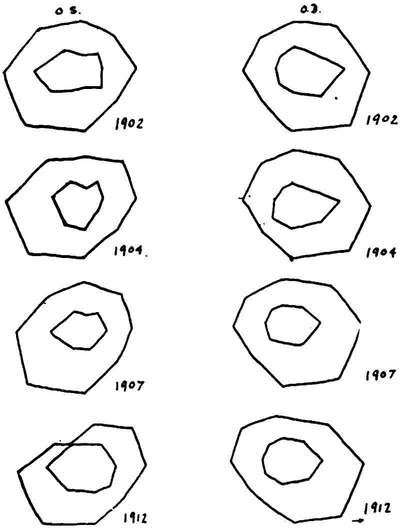

Fig. 16.—Fields of Case 16.

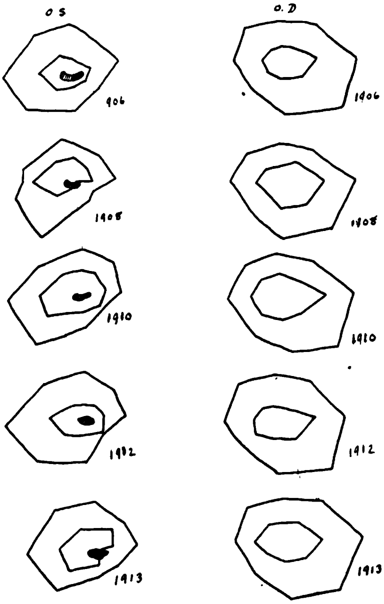

Fig. 17.—Fields of Case 17.

the deeper. In 1912, sharp attack of conjunctival irritation from the miotic. Controlled after a few weeks of local treatment.

*Remarks.*—Excellent result in a well-advanced case with marked vascular disease.

Case 17.—Woman, aged 52, first seen eight years ago. O. D. normal, commencing glaucoma cup in O. S.

O. D. + S. 0.50 D. ⊃ + C. 0.37 D. ax. 110° = 5/5.
O. S. + S. 0.50 D. ⊃ + C. 0.37 D. ax. 70° = 5/5.

Weak miotics prescribed for left eye. Two years later, retinal hemorrhage in macular region of left eye with some increase in glaucomatous symptoms at that time, the latter successfully controlled by increase in miotic strength. Progress most favorable during the past six years. Vision in O. S. still normal and field but little contracted—paracentral scotoma—the result of the old macular hemorrhage. The right eye has remained unaffected.

*Remarks.*—Disease of a hemorrhagic type perfectly controlled for a period of eight years by miotics. Operation would doubtless have been attended with hemorrhage and perhaps loss of the eye.

Case 18.—Woman, aged 65, first consultation December, 1907. This case permits of a comparison between the effects of iridectomy and miotics.

O. D., blind.
O. S. + S. 1.75 D. ⊃ + C. 0.75 D. ax. 105° = 5/5.

Deep glaucoma cup with atrophic nerve in O. D. Shallow-forming glaucoma cup in O. S. Typical chronic glaucoma. O. D. had been iridectomized by Dr. Noyes of New York ten years previously, but vision had gradually failed, and two or three years afterward complete blindness had ensued. Operative technic apparently perfect; large clear-cut iridectomy, lens clear. Miotic treatment directed to the left eye alone and has been persisted in for six and one-half years, with resultant normal vision, and with but a slightly contracted field to the nasal side. Tension, however, has become much more elevated and the cup deeper. The loss in field may be due in a measure to increasing cloudiness of the lens and to a very small pupil.

*Remarks.*—A perfectly formed iridectomy was apparently powerless to check the progress of the disease in the right eye, total blindness ensuing in two or three years, while miotics have maintained normal vision and an excellent field over a period of six years in the remaining eye, and this notwithstanding the advancing age of the patient.

Case 19.—Woman, aged 75, first seen Nov. 25, 1899. Failing vision past year. Shallow glaucomatous excavation in each eye, sclera rigid, anterior chamber shallow, slight haze of pupillary areas of both lenses.

O. D. + S. 1.5 D. ⊃ + C. 0.50 D. ax. 160° = 5/6.
O. S. + S. 3.5 D. = 5/7½.

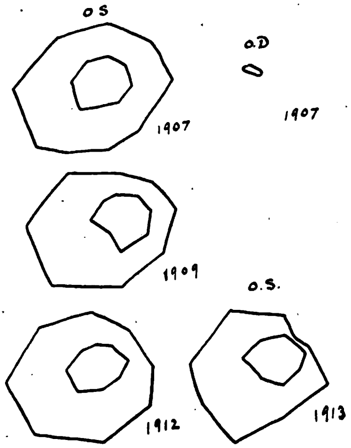

Fig. 18.—Fields of Case 18.

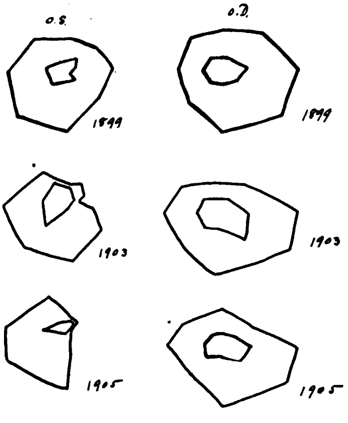

Fig. 19.—Fields of Case 19.

At end of 1 year:

O. D. V. = 5/6.
O. S. V. = 5/15

Some extension of glaucomatous process in O. S.

Iridectomy, with a view to lessening the glaucoma and to enlarging the pupils, so that more rays might enter the eyes, thought of, but the idea rejected, on account of age and feebleness of the patient.

When last seen, in June, 1905, a few weeks before the patient's death from apoplexy, O. D. V. = 5/12, O. S. V. = 5/35.

*Remarks.*—Result fairly satisfactory, loss of vision observed during six years being due quite as much to lenticular haze as to the glaucoma.

Case 20.—Woman, aged 69. First consultation six years ago. Deep glaucoma cups in each eye, with evidences of previous hemorrhages in each fundus. The right eye was blind, and though central vision in the left eye was normal, the field was cut almost to fixation. Miotics administered, and notwithstanding the advanced stage of the disease, vision is now 5/12 in the left eye. The field of vision embraces little more than fixation.

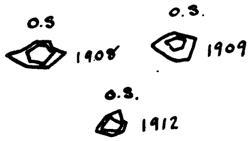

Fig. 20.—Fields of Case 20.

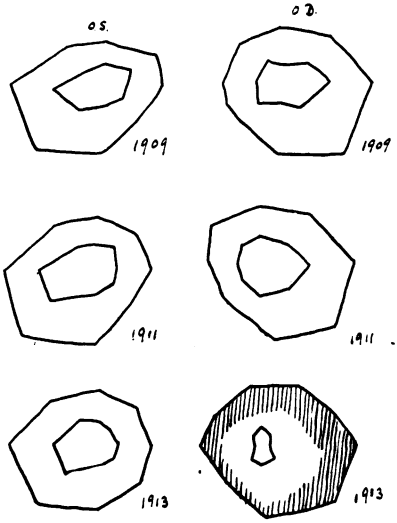

Fig. 21.—Fields of Case 21.

*Remarks.*—In view of the extreme contraction of the field in the left eye and depth of the cup and an unfavorable general condition, the result of the treatment in this case is remarkable.

Case 21.—Woman, aged 66, under observation six years. At first examination, commencing glaucoma cup in O. S. T+. O.D. normal. Slow increase in glaucoma in the left eye, but excavation still remains physiologic in the right. Tendency to conjunctival irritation. O. D. V. = 5/5, O. S. V. = 5/15

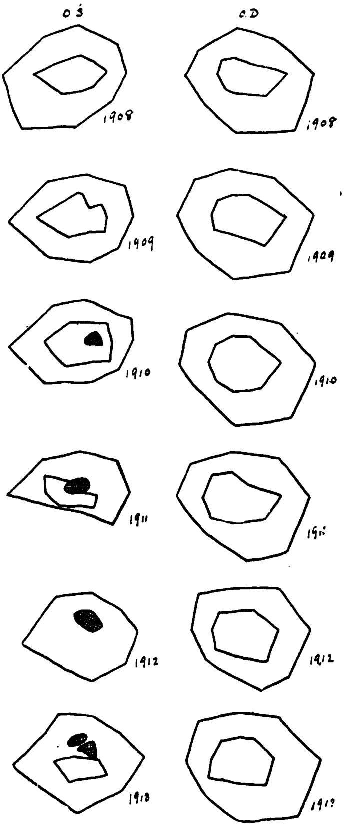

Fig. 22.—Fields of Case 22.

(poor vision in O. S. accounted for by localized haze in O. S. lens from an early traumatism).

Case 22.—Man, aged 53. When first seen, in September, 1908, had deep glaucoma cup in the left eye, with marked pallor of the nerve and evidences of degeneration in the retinal vessels. Tension markedly elevated. Right eye normal, with the exception of similar changes in the retinal vessels. Corrected vision 5/5 in each eye. Right field of vision normal, left concentrically contracted for form and color. Miotics prescribed, and treatment directed to allay further vascular mischief. Abstinence from tobacco insisted on but not followed. Normal vision maintained for two years, when hemorrhagic retinitis appeared in left eye, reducing vision to 3/80. After persistent care in the general treatment and the use of miotics, vision rose to 5/12, at which point it is still maintained. The right eye is still free from glaucoma, with normal vision and field.

*Remarks.*—Monocular glaucoma of a hemorrhagic type held in check for six years by miotics. Fellow eye still free from the disease. By reason of the advanced changes in the vessels in each eye, any form of operation would probably have been followed by severe hemorrhage, with loss of the eye.

Case 23.—Woman, aged 59, first seen six years ago. Right eye blind with chronic glaucoma. Vision in this eye had begun to fail when patient was 47 years of age, and blindness had ensued without treatment in four years. The vision of the left eye had always been poor, the eye having squinted in childhood. Corrected vision equaled 5/35 at time of first examination, and the field was normal. Note made of sclera being rigid, but no glaucoma cup present. Miotics prescribed, and continued without interruption for six years, with present vision of 5/35 and normal field. Shallow glaucoma cup.

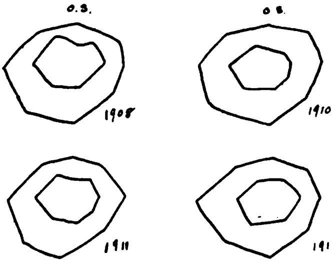

Fig. 23.—Fields of Case 23.

*Remarks.*—Without treatment, right eye became blind from glaucoma in four years; normal vision still maintained in the fellow with but little advance of the glaucomatous process over a period of six years.

Case 24.—Man, aged 42, Hebrew, at first consultation, Jan. 10, 1908, complained of dilatation of right pupil and of misty vision and occasional halos in that eye for six months previously. Myopia from early youth. Examination revealed dilated pupil, a slightly steamy cornea and a forming glaucoma cup, in the right eye. The retinal veins were also dilated, especially the inferior, and near the disk there was a small flame-shaped hemorrhage. The left eye was normal.

O. D. V. — S. 8. D. ⊃ —C. 1.75 D. ax. 35° = 5/6 partly.
O. S. V. — S. 8. D. ⊃ — C. 2.5 D. ax. 150° = 5/5.

Miotics in both eyes advised and this opinion confirmed by Dr. de Schweinitz, in consultation. This treatment has been carried out with great regularity for the past six years, and though the cupping has extended itself so that it has now assumed an appearance typical of glaucoma, vision and field of vision are normal. The left eye has remained normal throughout.

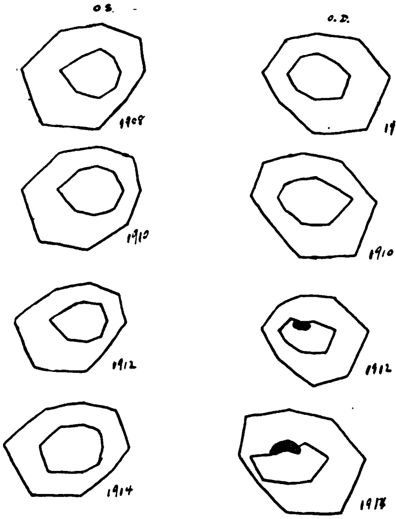

Fig. 24.—Fields of Case 24.

*Remarks.*—Although predisposed by racial characteristics to glaucoma, and to a hemorrhagic type of the disease, miotics have controlled the disease for six years, and vision is still normal. In consequence of the vascular changes, an early operation would doubtless have occasioned intra-ocular hemorrhages and precipitated blindness.

Case 25.—Woman, aged 55. First consultation Dec. 8, 1903. Commencing glaucoma cups in each eye. T +.

O. D. — S. 7.5 D. ⊃ — C. 2.5 D. ax. 100° = 5/7½.
O. S. + S. 0.25 D. ⊃ — C. 0.50 D. ax. 120° = 5/5.

Seen at yearly intervals and miotic treatment persisted in during the intervals. When last seen, in 1908, with correction O. D. V. = 5/12, O. S. V. = 5/6. Excavation in each eye more extensive and tension higher. Left field of vision contracted; right scotomatous.

*Remarks.*—But little loss of vision in the five years she was under observation. If miotics are persisted in, she will doubtless retain good vision for many more years.

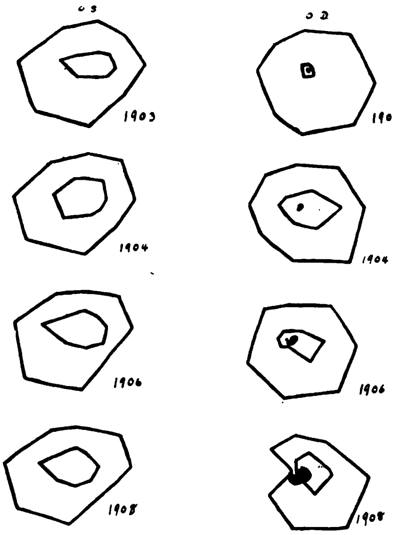

Fig. 25.—Fields of Case 25.

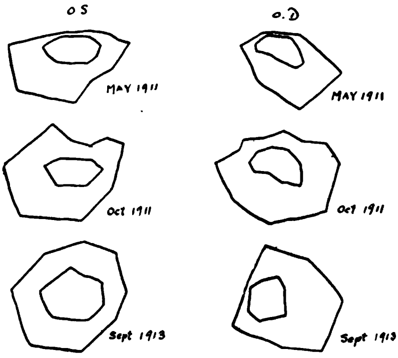

Fig. 26.—Fields of Case 26.

Case 26.—Woman, aged 26. First consultation May, 1911. This patient was sent to me in consultation by Dr. W. P. Reaves of Greensboro, N. C., on account of failing vision from a chronic glaucomatous process in each eye. At the time of examination:

O. D. V. — S. 1. D. ⊃ + C. 1.75 D. ax. 15° = 5/12 partly.
O. S. V. — S. 0.75 D. ⊃ — C. 1.25 D. ax. 75° = 5/12 partly.

Both optic nerves were semiatrophic and deeply cupped. Tension equaled + 1 in each eye. The upper halves of each field of vision were much contracted. Miotics were prescribed, and six months later the vision in each eye had risen to normal and the fields were larger. Through the courtesy of Dr. Reaves, this patient has reported semiannually and an improvement was noted at each visit until that made in September last, when it was found that O. D. V. had decreased to 5/9 and O. S. V. to 5/7½. The field of the right eye showed a decided loss in the nasal half. Tonometer registered 33 mm. Hg in each eye. Elliott operation advised but refused.

*Remarks.*—Miotics seemed to control the glaucomatous process in this patient, in whom the disease appeared at a most unusually early age, for almost two years. Pilocarpin is still employed and the vision is still at the same degree as last noted.

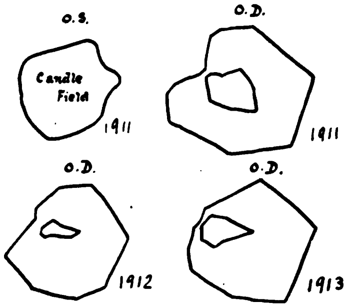

Fig. 27.—Fields of Case 27.

CASE 27.—Man, aged 74. First consultation Feb. 24, 1911. Progressive failure in vision of left eye for one year—no treatment. Still sees well with right eye. Subject to inflammatory rheumatism for past twenty years. Deep glaucoma cups with increased tension in each eye. O. D. V. = 5/6, O. S. V., hand movements at 1 foot. Deep cut in nasal field of right eye. Miotics administered in usual manner and continued with great fidelity. Corrected vision now equals 5/7½, the slight loss being attributable perhaps to increasing haziness of lens. Field but little more compromised than at first visit.

*Remarks.*—Vision is still good, despite advanced age of patient and the progress of the disease, even at time of first examination.

Case 28.—Woman, aged 81. First consultation Oct. 13, 1908. Falling vision past three years, especially in O. D. Deep glaucomatous cups in each eye. Lenses somewhat hazy. O. D. V. = counting fingers at 6 inches eccentrically. O. S. + S. 2. D. = 5/12. Field contracted, paracentral scotoma.

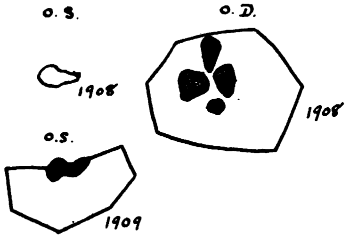

Fig. 28.—Fields of Case 28.

Pilocarpin persisted in for the two years she was under observation, with a resultant vision of 5/22 in the left eye, loss of upper half of field.

*Remarks.*—Preservation of vision for two years and perhaps longer in a so-called "desperate" case.

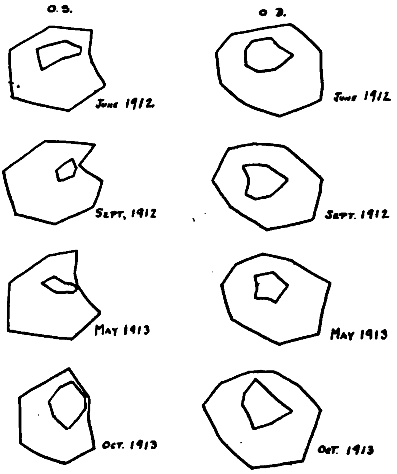

Fig. 29.—Fields of Case 29.

Case 29.—Woman, aged 77. First consultation June 1, 1912. Vision failing in each eye for several years, said by several oculists to be due to cataract. Examination showed pronounced glaucoma cups in each eye, with some lenticular opacities.

O. D. — S. 0.37 D. ⊂ + C. 1.25 D. ax. 75° = 5/6.
O. S. — S. 0.50 D. ⊂ + C. 1.75 D. ax. 100° = 5/9.

Miotics prescribed and despite lenticular haze vision and field still remain, after the lapse of almost two years, as at first examination.

*Remarks.*—This excellent result from the miotics attained in a patient of advanced years with marked lenticular changes.

Case 30.—Man, aged 81. First consultation Feb. 5, 1913, on account of failing vision for some years previously. Chronic glaucoma had been diagnosed two and one-half years previously and iridectomy advised. Operation was, however, refused and miotics had been prescribed, which had been used at irregular intervals. Examination revealed deep glaucoma cups with same optic nerves in each eye. Considerable haze of the nuclei of both lenses.

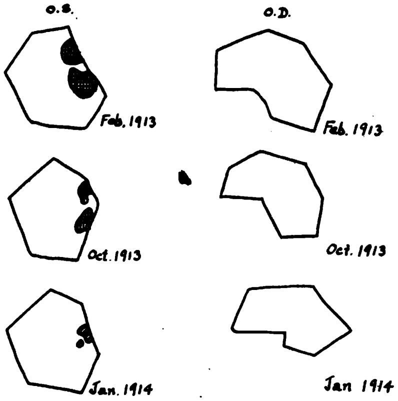

Fig. 30.—Fields of Case 30.

O. D. V. = 3/60, not improved.
O. S. V. + C. 1.5 D. ax. 180° = 5/100.

Miotics prescribed, bringing O. D. V. = 5/60 and O. S. V. = 5/45 and maintaining this degree of vision in each eye until the present.

*Remarks.*—A most excellent result from the miotics, when one considers the age of the patient and the advanced stage which the disease had attained when full miotic treatment was inaugurated.

Case 31.—Man, aged 72, a patient of Dr. Cheney of Boston, was seen in consultation April 1, 1908. At that time, deep glaucomatous excavation in O. D. and a forming one in O. S. O. D. field contracted to nasal side and several large paracentral scotomas. O. S. field normal. Miotics ordered, retaining central vision in the right eye for two years, when useful vision in that eye gradually disappeared in consequence of a slowly contracting field and the formation of a complete ring scotoma. At this time there remains but a small field up and out. O. S. vision and field of vision are both normal.

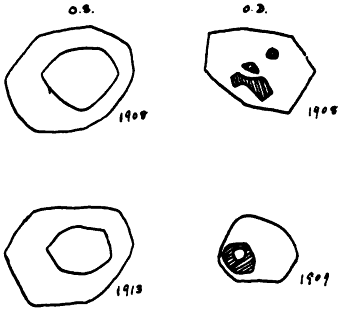

Fig. 31.—Fields of Case 31.

*Remarks.*—Vision maintained in O. D. for two years, despite the advanced process of the disease when treatment was first inaugurated. Vision still normal in left eye after a period of six years.

Twenty-First and Chestnut Streets.

# MEMORANDA

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# INTERNATIONAL STANDARD FOR TESTING VISION AND STANDARDIZING OTHER VISUAL TESTS *

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The cards of test-letters in common use are admirably adapted to the subjective determination of errors of refraction; but as a test for visual acuity they probably afford the poorest and most inexact standard on which any scientific observations are now based. As a standard for visual acuity, test-letters belong to the stage of mental and scientific development when the standard for length was "three full grains of barley corn taken from the middle of the ear," or "the distance a man can walk between sunrise and sunset."

As a help in the subjective measuring of refraction they are superior to any substitute that has been proposed and are not likely to be given up. But as a scientific standard they possess two essential defects:

1. The different letters when made as uniform as possible are visible from very different distances. This makes them unsuitable for a scientific standard.

2. They can be readily committed to memory by all who are sufficiently familiar with them to make them a convenient test. This lessens their value as a practical test.

For these reasons test-letters should be standardized before they are used as a basis for records of visual acuity, and they should not be used when testing visual acuity is the only purpose to be served.

Every one who has attempted to establish a scale of test-letters has been struck by their differences in visibility. When Snellen attempted to make letters uniform by using a block-letter in which the lines

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

and minimum spaces should have a uniform width, subtending a 1-minute angle at the standard distance, each letter to occupy a 5-minute square, he rejected as unsuited to the purpose the letters I, J, M, Q, W and X. Green carried the process farther, rejecting twelve letters and using fourteen. Oliver used only seven, but he still admitted letters that varied as much as 40 per cent. in visibility. The logical conclusion was reached only when a single set figure was adopted as the test, as in Snellen's optotypes for the illiterate published in 1873 (Fig. 1 *A*).

Before this section at the Nashville meeting in May, 1890, I proposed the simple form of the incomplete square, subtending an angle of 3 minutes (Fig. 1 *B*); in 1889 Landolt had suggested the broken ring (Fig. 1 *C*), and at Naples in 1909 the International Ophthalmological Congress adopted the broken ring as an international standard test for visual acuity, the most important official action that has ever been taken by an international ophthalmological gathering. Either one of these tests furnishes an exact measure for visual acuity, so far as visual acuity is capable of exact measurement, and one enormously superior to any furnished by letters, numerals or any series of conventional figures.

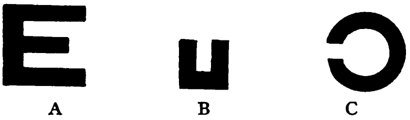

Fig. 1.—*A*, Snellen's optotypes. *B*, Jackson's incomplete square subtending an angle. *C*, Landolt's broken ring.

The difficulty of using a series of differing figures for a scientific test has been encountered by every one who has attempted to arrange a series of test-type, but few have appreciated the impossibility of an accurate standard of visual acuity resting on such a basis. In 1885 Dennett[1] read before the American Ophthalmological Society a paper on test-type in which this unsuitability of letters to furnish a standard test was well brought out. More recently it has been illustrated by the work of Gebb and Löhlein.[2] Dennett took the block-letters of the forms used in Monoyer's test-card, and found that letters 25 mm. in height could be recognized at distances that varied from 10.3 meters for the B and S, to 17.7 meters for U. He analyzed the differences of visibility and proposed and arranged test-cards in which the letters of each line should all be visible at nearly the same distance. To show how different such a test is from those we are accustomed to rely on, a line from one of these cards is reproduced in Figure 2.

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[1] Dennett: Tr. Am. Ophth. Soc., 1885, iv, 133.

[2] Gebb and Löhlein: Arch. f. Augenh., 1910, lxiii, 69.

So far as I know Dennett's tests have never been extensively used. While they would be superior to any other published series of test-letters for the scientific testing of visual acuity, they are not quite equal to the international standard for this purpose, and are distinctly inferior for the other purpose for which test letters are employed, the subjective determination of errors of refraction.

It would be absurd to spend time arguing the unfitness of letters as ordinarily printed on our test-cards for making scientific measurements, if they were not almost universally employed for that purpose. To use a tape measure that would spontaneously contract to 20 inches or stretch to 36 inches would be an absurdity of the same order as to use as equivalents letters, some of which can be just recognized at 20 feet and others at 36 feet, calling them all 20-foot letters.

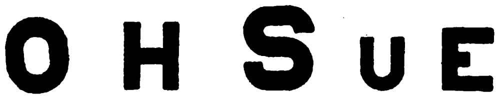

Fig. 2.—One of Dennett's lines of test letters. All about equally visible at 20 feet.

Such is the variability of letters of a single style used on a single card. The recognized forms of letters, even of capital block-letters, to be found in a well-equipped printing establishment, run into the hundreds. Three or four fairly distinct types of block-letters are in use on our test-cards. The differences of visibility vary with all these different forms. One firm has put out test-cards on the five-minute basis and others on a four-minute basis. The letters on the latter, although only four-fifths the height, can be seen quite as far as those on the former, because they are of simpler form. The block-letter U, if made as at *A* in Figure 3, can be recognized 50 per cent. farther than if made as at *B* in Figure 3. When to these differences in the letters of the roman alphabet are added the myriad differences of the different alphabets of the world, the ***reductio ad absurdum*** of testing visual acuity by letters becomes complete.

In contrast with this take the mathematical exactness and constancy of the international standard test. If one always used the same test-card and could have the same letter or letters of equivalent visibility on every line of the card, it would be possible to obtain records of practical accuracy for individual use. As this is not done, when different cards are used, and as different lines contain letters of different visibility, the test-letters fail us even in this practical service. I have a test-card in which the letters are so chosen that the 6-meter line is quite as easy to read, perhaps is more often read correctly at the same distance, than the 7.5-meter line on the same card.

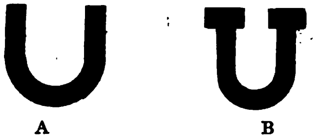

Fig. 3.—Different forms of the same letter inscribed in squares of the same size, subtending an angle of five minutes at about 30 feet.

Our test-cards become brown with age, or worn out, and often are not exactly replaceable. I doubt if I could get now, without having them especially made, samples of any of the test-cards used in my first five years of practice. There are advantages in using different test-cards in testing the two eyes, or in testing the same patient on successive days, and as the different cards have different series of letters to be recognized at the same distance, the practical result is that all records of visual acuity made with test-letters have a very indefinite significance, even in the practice of one surgeon. When it comes to comparing the work of different men, especially in different parts of the world, the practical value of records of such visual acuity is greatly impaired. If the 6-meter line is easier to read than the 7.5-meter line, what do we learn when told that at one visit the patient read most of the 7.5-meter line, and then under treatment he read a week later all of the 6-meter line?

So much for the first objection to test-letters. The second objection is of equal practical importance. It has not been very rare for men required to pass visual tests for a government or railroad service to obtain a copy of the test-card used and commit the letters to memory and thus pass the usual examination without possessing the required visual acuity. Many ingenious contrivances have been devised to prevent such evasions of the test. William Thomson arranged radiating lines of test-letters on a circular card, only one of which lines could be exposed at any one time. Yet a person obtaining a card and memorizing the lines could repeat the smallest letter from his recognition of the largest. Grow[3] devised a test-card which he called unlearnable, having sixty letters all of the standard 20-foot size, of which only a single line was exposed in such a way that the observer himself could not know which line was in use except by reading it.

Burch[4] wrote, "It is astonishing how soon the exact description of a test-card becomes known if many people from the same neighborhood are tested." For the confusion color-test of vermilion letters on a green ground, he advises that the examiner keep paints of the right colors, and paint in new letters for any individual case. The late Dr. George C. Harlan used to tell of a schoolboy who, tested one day with an ordinary test-card of Snellen letters, was able next day to repeat the whole card correctly from memory. The caution sent out with cards for testing schoolchildren, to keep the test where the children will not have a chance to see it, is quite necessary. Probably all of us know without looking at the card the succession of letters on any test-card that we have used much, and the element of memory renders a patient's answers unreliable after a letter has once been recognized, so far as that letter is concerned.

So long as the vision has improved by a change of glasses and progressively smaller or more difficult letters can be read, the ordinary letter or numeral test-card serves its purpose most admirably; but when it is used simply to ascertain the maximum visual acuity, the chance of remembering the letter, instead of seeing it, is a serious drawback. This chance is not confined to letters or numerals. It applies to all cards of test-figures that cannot have their directions or relations changed at will.

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[3] Grow: Ophthalmology, 1911, vii, 195.

[4] Burch: Ophthalmoscope, 1912, x, 320.

Here lies the great advantage of the incomplete square or the broken ring. As was pointed out twenty-five years ago by Landolt, and a few months later independently by myself, if the card on which one or more of these tests are symmetrically placed is simply turned, it becomes impossible for the person tested to know, except by sight, in what direction the incomplete side is placed. By using a single broken ring or a symmetrical group of them printed on the center of a symmetrical card, the same card can be used for any number of repetitions of the test, without giving any hint of the correct answer, except by vision sufficient to recognize the figure.

This manner of using such a test makes it impossible to have a single card with a whole series of broken rings of different sizes printed on it. But one size can be placed on each card. The same amount of cardboard cut into the proper pieces can be made to furnish an equally complete series. When a large number of persons are to be tested, the best plan is to use a single test of a certain standard size. This is to be looked at first from or beyond the standard of distance. Then the person to be tested should gradually approach the card, until the direction of the break is recognized.

For instance, in testing a class of schoolchildren, the test should be shown to the whole class and its use explained. Then the children should be sent beyond the standard distance and each one made to approach until the break in the ring is recognized. If we take the standard distance of 5 meters, recommended by the International Ophthalmological Congress, and mark it off on the floor in 0.5-meter spaces, or double this distance and mark it in whole meters we can, at a glance, ascertain the visual acuity in tenths. It is true that theoretically the variable distance introduces a minute inaccuracy through varying divergence of the rays from the test object; but this theoretical inaccuracy has no practical importance, and is so extremely minute in comparison with the inaccuracies that we have every day tolerated in connection with the use of test-letters that it may well be disregarded. What is meant by a symmetrical group of broken rings is illustrated in Figure 4.

The rings may be arranged in squares as 1, 4, 9, 16; or any number may be arranged in circular form. I believe that as its advantages are appreciated, the international standard test will come into universal use for the simple testing of visual acuity.

The international test, however, does not so directly help us in the problems of the consulting-room in practically determining and recording the visual acuity obtained by use of test lenses, a very important part of our daily work. In this work series of test-letters arranged in graduated sizes will hold their own against any other method of testing visual acuity that has yet been suggested.

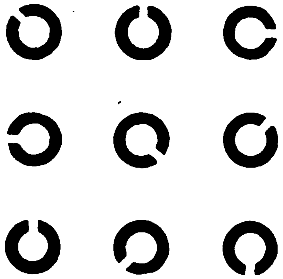

Fig. 4.—International test. With normal vision, the direction of the break in the ring can be recognized at 5 meters.

With our ordinary cards of test-letters the practical accuracy of the international standard can be easily attained by the very simple expedient of numbering each line of letters by its equivalent value as compared with the international standard. For instance, instead of calling the 5-meter line a 5-meter line or a 16½-foot line, if it contains some of the more difficult letters, like B or S, number it 1, as expressing full vision according to the international standard. At the same time if half the letters on it can be read by an eye having vision of only 9/10 by the international standard, also indicate this on the card; half the letters equal 9/10, all the letters equal 1. If one or two letters can be recognized by a person having 8/10 vision, that fact might also be indicated; but practically it will usually be best to give only the two values; one value for a certain portion of the letters, as 1/2, and the other the acuity indicated by reading every letter on the line. In a word, we must standardize our cards of test-letters. When this has been done it is just as easy to read the standard of visual acuity indicated from the card used as to read the old meters or feet printed with each line to indicate the distance at which it was supposed letters of that size should be visible.

With the test-card standardized, we can use any arrangement of letters we choose, and as many different arrangements as we choose. We can employ one card one day and another the next day or the next month, or after twenty years, and still know that the comparisons of visual acuity are accurate. Each nation may test vision by cards suited to its own convenience. Illiterate test-cards of any sort may be employed, and if properly standardized the results will be perfectly comparable.

This matter, simple as it is, is one of great practical and scientific importance. It is entirely worthy of the careful attention of every association of scientific ophthalmologists. To standardize the cards already in use, and to bring the matter properly to the attention of those who publish such cards, it is suggested that this section appoint a committee, and that every member of this Section should cooperate with that committee to secure the best possible standardization of all test-cards in common use.

Majestic Building.

# MEMORANDA

## MEMORANDA

# THE ETIOLOGY OF PHLYCTENULAR OPHTHALMIA

## IS TUBERCULOSIS REALLY AS IMPORTANT A FACTOR IN THE CAUSATION OF THIS DISEASE AS IS NOW COMMONLY TAUGHT? *

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BALTIMORE

Few questions in ophthalmology have been more discussed in recent years than the etiology of phlyctenular ophthalmia. This is accounted for by the fact that during this period the theory has been advanced, and has been supported by many eminent ophthalmologists, that phlyctenulosis is a tuberculous manifestation.

The advocates of this doctrine unceremoniously reject the older view of the etiology of this affection, which led especially German writers on diseases of the eye to designate it as ophthalmia eczematosa, and, in conformity with their convictions, urgently commend in its treatment the employment of tuberculin, as the most efficacious remedial agent at our command. This being the case, it is evident that the true nature and causation of phlyctenular ophthalmia is not merely an academic question, but is one of very practical importance; for, after all, if it is really *not* tuberculous, the use of tuberculin in its treatment is sure to do more harm than good, and is most distinctly contra-indicated.

Let us, then, consider the evidence which has been advanced in support of the new doctrine, and try, impartially if we can, to determine its import. And here it may be premised that the advocates of the tuberculous theory no longer contend that the conjunctival or corneal phlyctenules are examples of true ocular tuberculosis, since they are compelled to admit not only that they never contain tubercle bacilli, and that tuberculosis has never been produced in animals by inoculating them with the contents of the phlyctenules, but that histologically the phlyctenules bear no resemblance whatever to true tuberculous tissue. What they do maintain is that phlyctenulosis is a pseudotuberculous or paratuberculous affection, caused by the action of the toxins of tubercle bacilli present somewhere in the body.

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

In support of this belief they cite:

1. The frequency—from 70 to 90 per cent., according to different investigators—with which the subjects of phlyctenulosis give a positive reaction to diagnostic tests with tuberculin, the von Pirquet test being chiefly relied on.
2. The efficacy of tuberculin in the treatment of phlyctenulosis, and especially its efficacy in controlling chronic cases and those showing a tendency to recurrence.
3. The development of phlyctenular conjunctivitis and keratitis after the application of tuberculin to the eye, and after other diagnostic tuberculin tests.
4. The occasional occurrence in phlyctenular ophthalmia of focal reaction following diagnostic or therapeutic injections of tuberculin and after the von Pirquet test.
5. The chronicity and tendency to recurrence of the disease, its predilection for the poorly nourished and for those in whom the existence of the so-called strumous or scrofulous diathesis is manifest, and the glandular complications which frequently accompany it.

Before taking up this summary of the evidence advanced in support of the tuberculosis theory, let us, for a moment, consider the significance of the admitted non-existence of tubercle bacilli in the phlyctenules, and, further, the present assertion that phlyctenulosis is a pseudotuberculous affection, due to the toxins of the tubercle bacillus.

Dr. John W. H. Eyre,[1] Hunterian professor, and examiner in bacteriology, Royal College of Surgeons, in his Hunterian lecture on "Tuberculosis of the Conjunctiva," makes this very emphatic pronouncement:

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[1] Eyre, John W. H.: Lancet, London, 1912, i, 1319.

> That many of those subject to phlyctenules give evidence of the existence of tuberculous infection in some portion of their tissues is no evidence that the conjunctival lesion is also tuberculous, and until some responsible observer has demonstrated the presence of the tubercle bacillus in an extended series of phlyctenules, I see no valid reason for regarding phlyctenular conjunctivitis, *per se*, as a form of tuberculosis of the conjunctiva.

Dr. L. V. Hamman, chief of the Phipps Tuberculosis Dispensary of the Johns Hopkins Hospital, and one of the authors of Hamman and Wolman's authoritative work, "Tuberculin in Diagnosis and Treatment," in conversation with me, has expressed a similar view. The substance of his statement was that if the phlyctenules are a tuberculous manifestation they certainly *sometimes* should show the presence of the tubercle bacillus; otherwise the theory that they are of tuberculous origin is without warrant. He also drew attention to the fact, as bearing on the contention that the phlyctenules are a pseudotuberculous manifestation due to the toxins of the tubercle bacillus, that this view of the action of toxins is rapidly losing ground, and that very many pathologic lesions formerly accounted for in this way have since been proved to be due, not to the action of toxins, but to the presence in these lesions of the bacteria themselves.

Let us now take up in order the evidence on which the doctrine of the tuberculous nature of phlyctenular ophthalmia rests.

*1. The frequency with which the subjects of phlyctenulosis give a positive reaction to the von Pirquet and other diagnostic tuberculin tests.*

To this evidence the advocates of the doctrine can hardly attach much weight, since Hamburger of Vienna and others have shown that about the same percentage of positive reactions to the von Pirquet test is encountered in healthy persons as has been found in those with phlyctenulosis. On this point Eyre, already quoted, says:

> I have come to regard a positive reaction to these [tuberculin skin] tests when occurring in an individual more than 1 or 2 years old as quite useless for purposes of diagnosis.

And, just as emphatically, Hamman and Wolman[2] say:

> A positive reaction [to von Pirquet's test] is of considerable value in diagnosis during the first year of life, of less during the second, of little in childhood, and of none in adult life.

It is, perhaps, hardly necessary to remark that, in the absence of definite evidence of clinical tuberculosis, no competent and conscientious physician would feel himself warranted in employing tuberculin therapeutically, simply because the von Pirquet test elicited a positive reaction.

*2. The efficacy of tuberculin in the treatment of phlyctenulosis, especially in chronic cases and in preventing recurrent attacks of the disease.*

It has not been possible for me to make even an approximately thorough study of the extensive literature of the past few years dealing with the theory of the tuberculous nature of phlyctenular ophthalmia. Two important papers published in this country, however, which together include a summary of the results obtained in ninety cases of phlyctenulosis treated with tuberculin, seem to afford fairly conclusive evidence as to the efficacy of this treatment.

Fifty of these cases were reported by Dr. Richard J. Tivnen[3] of Chicago in a paper read before the Section on Ophthalmology of the American Medical Association in 1911. It is to be regretted that, though the author in the tabular report of these cases heads a column "Therapeutic Duration of Treatment," he gives under this heading only the date when the treatment was *begun,* and does not in a single instance mention its *duration.* Measurably to offset this omission, however, he states elsewhere that the shortest period of treatment of any case was "about three weeks," and the longest six months.

Turning to the column in which the duration of the disease before treatment was begun is given, we find that eleven of the fifty cases had existed three months or less. Of these only two had lasted this long, five were of two months' duration, one of six weeks, one of one month, one of two weeks, and one had existed only two days. None of these cases can be regarded as chronic in character, and that not one of them, not even that of two days' duration, was cured in less than "about three weeks" seems to afford but poor evidence of the value of the treatment employed. Indeed, when it is borne in mind that in addition to the administration of tuberculin the usual ocular treatment, such as yellow oxid of mercury, atropin, etc., was employed, that constitutional measures, such as regulation of the diet and hygienic conditions, were not overlooked, and further that "tonics" were administered, it is difficult to escape the conviction that the tuberculin was a definite handicap to the other very proper therapeutic measures, and that the results would almost surely have been more satisfactory had it not been employed. It may be added, as of interest, that in only six of the cases (12 per cent.) was there a history obtained of either active or latent tuberculosis.

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[2] Hamman and Wolman: Tuberculin in Diagnosis and Treatment, p. 126.

[3] Tivnen, Richard J.: Tuberculin as a Diagnostic and Therapeutic Agent in the Treatment of Conjunctivitis Eczematosa (Phlyctenular Conjunctivitis and Keratitis), THE JOURNAL A. M. A., Dec. 9, 1911, p. 1886.

The other paper referred to was by Davis and Vaugan,[4] published in September, 1912. Like Dr. Tivnen, they also head a column in their tabular summary of forty cases of phlyctenular ophthalmia treated with tuberculin "Therapeutic Duration of Treatment," but, under this head, mention only when the treatment was begun. For this they make partial amends by stating, in another part of their paper, that the duration of treatment varied from "a few weeks" to eight months.

It is interesting to note that of the forty cases the result of the von Pirquet test was positive in only twenty-seven (67.5 per cent.), and was negative in thirteen cases (32.5 per cent.), hardly as large a percentage of positive results as might be expected in the same number of healthy persons.

Turning to their column showing the duration of the disease before treatment, we find no less than fourteen cases (35 per cent.), in which this is given as two months or less. Of these the duration of four was two months, three "off and on" for two months, four one month, one two weeks, and two one week. The same comment applies to these cases as to the acute cases of Dr. Tivnen's table. It seems remarkable that not one of these comparatively recent cases was cured in less than "a few weeks." That their results were even less favorable than those of Dr. Tivnen is doubtless accounted for by the fact that they employed no constitutional treatment but tuberculin and "but little local treatment," atropin having been used in but two cases. It may be added, as worthy of note, that but *one* of their cases showed clinical signs of active or inactive tuberculosis—tuberculous glands of the neck being present in this case.

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[4] Davis, A. Edward, and Vaugan, Harry: Phlyctenular Conjunctivitis and Keratitis, with Special Reference to Etiology and the Value of Tuberculin as a Diagnostic Agent, Ophth. Rec., 1912, xxi, 476.

One of the best-equipped and most careful students of ocular tuberculosis in this country, Dr. George S. Derby of Boston, has probably had as large an experience with tuberculin in the treatment of phlyctenular ophthalmia as any one in America, and he has expressed his views very plainly as to its value in this condition. In discussing Dr. Bruns' paper on "Phlyctenular Ophthalmia and Its Etiology," in the Section on Ophthalmology of the American Medical Association, at Atlantic City, in June, 1912, he said:

> I have had considerable experience with tuberculin in the treatment of these [phlyctenulosis] cases during the past five years, and I feel doubtful whether any of the cases have been much benefited by it.

And, again, in discussing Dr. Will Walter's paper, "Phlyctenular Ophthalmia and Episcleritis," before the same Section, in 1913, he said:

> The use of tuberculin in phlyctenular disease is not often necessary. Most of these patients recover without tuberculin. Even though tuberculin is used, *it is often extremely difficult to prevent repeated attacks in obstinate cases.* . . . It is not nearly so important as general hygiene.

I have taken the liberty of italicizing this observation of Dr. Derby as to the inefficacy of tuberculin in preventing repeated attacks in obstinate cases of phlyctenulosis, for it is to be borne in mind that its efficacy in accomplishing just what Dr. Derby's experience shows that it does not accomplish has been cited as one of the proofs of the tuberculous nature of phlyctenular ophthalmia.

*3. The development of phlyctenular conjunctivitis and keratitis after application of tuberculin to the eye and after other diagnostic tuberculin tests.*

On this point Hamman and Wolman[5] say:

> In several thousand [ocular] instillations [of tuberculin] we have had but two untoward results, one patient developing phlyctenular conjunctivitis, which subsequently completely healed, the other, an episcleritis, which also healed, and which we are not sure was due to the test.

In my own mind there is little doubt that had the instillations in these several thousand eyes of persons whose condition seemed to demand a tuberculin test consisted of a 2 per cent. solution of silver nitrate or copper sulphate, a considerably larger number of phlyctenular cases would have developed; for every one of large experience in the management of ocular maladies has seen cases of phlyctenular keratitis or conjunctivitis which were provoked by the application of unduly severe remedies to the eye, and I can recall others in which the attack followed a slight traumatism, such as an abrasion of the cornea or bulbar conjunctiva.

Some who teach the tuberculous doctrine of phlyctenulosis hold that "some external, local stimulant is necessary to bring out the phlyctenules." If this view is correct, why, I would ask, should not the tuberculin employed in the Calmette test, acting simply as a stimulant or irritant, occasionally produce this effect?

It is, perhaps, not so easy to explain satisfactorily the development of phlyctenules after the skin or subcutaneous tuberculin tests. It is to be said, however, that this is an unusual occurrence, and it may be questioned whether it ever happens, except in persons whose systemic condition is such that an outbreak of phlyctenulosis may be regarded as imminent. Under such circumstances it is readily conceivable that any added constitutional disturbance, such as the systemic effect of tuberculin, or that which might follow the ingestion of unwholesome food, may turn the scales, and precipitate the ocular inflammation, just as, under like circumstances, the application of any irritant to the eye may do.

*4. The occasional occurrence in phlyctenular ophthalmia of focal reaction following diagnostic or therapeutic injections of tuberculin or after the von Pirquet test.*

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[5] Hamman and Wolman: Tuberculin in Diagnosis and Treatment, p. 209.

Dr. Tivnen in his paper, already quoted, mentions the occurrence of focal reaction after the von Pirquet test in four of his fifty cases of phlyctenulosis, but describes the reaction in three of these as being "slight." Drs. Davis and Vaugan in their forty cases mention three instances of focal reaction, one of these being "slight." If we interpret, and this seems not unwarrantable, the three "slight" reactions of Tivnen's series and the one of Davis and Vaugan's as "doubtful," we have in the combined series of ninety cases but three definitely positive focal reactions—a percentage of only 3.33. Although I am aware that it is not in accord with the commonly accepted view, I cannot persuade myself that any constitutional disturbance comparable to that produced by the von Pirquet test would not have influenced unfavorably the ocular inflammation quite as often and quite as markedly as happened in these ninety cases after the tuberculin test.

It is worthy of remark that in neither of the papers in question is there mention of focal reaction having resulted, in a single instance, from the *therapeutic injection of tuberculin.* If such reactions had been observed, it seems most improbable that they would not have been recorded as evidence in favor of the authors' views regarding the etiology of phlyctenular ophthalmia; if no such reactions occurred, the circumstance is certainly of much significance.

*5. The chronicity and tendency to recurrence of phlyctenular ophthalmia; its predilection for the poorly nourished and for those in whom the existence of the so-called strumous or scrofulous diathesis is manifest, and the glandular complications which frequently accompany it.*

As is universally recognized, phlyctenular ophthalmia is essentially a disease of childhood. In the exceptional cases encountered in adults it will generally be found that the affection first manifested itself in childhood, and has recurred from time to time in after years. These, in my experience, are the cases in which the "scrofulous" diathesis, the glandular complications, and not infrequently, I am willing to admit, definite clinical signs of tuberculosis, are found. On the other hand, the much larger number of cases encountered in childhood, which commonly respond promptly to treatment, and which, if they recur at all, do so only during childhood, do not show, or, at all events, very rarely show, any signs of scrofula or tuberculosis. These are the cases, I contend, which must be regarded as typical, and it is from the study of these cases that we must draw conclusions as to the etiology of the affection, and not from the exceptional and atypical cases encountered in adult life.

What, then, does the study of these cases of childhood show? In the first place, it may be said that the children brought to our clinics with phlyctenulosis are usually not puny and ill nourished; on the contrary, they are commonly well nourished, and not infrequently have ruddy cheeks. Nine times in ten we find, as evidence of systemic disorder, in addition to the ocular inflammation, facial eczema, eczema perhaps elsewhere, and very often an eczematous nasal catarrh. On inquiry it will be learned that they are fed on nearly everything that a child should not eat, and that they are much too closely confined in rooms or dwellings that are thoroughly insanitary. Further, it will often be found that their tongues are furred, their bowels in a torpid condition, and that their stools are foul-smelling.

Perhaps I have already said enough to make it evident that I am a firm believer in the view formerly held by most ophthalmologists, and especially by German ophthalmologists, that phlyctenular ophthalmia, certainly as it is encountered in children, is an ocular eczema, and that it has just as much, and just as little, to do with tuberculosis as has the facial eczema which is its usual accompaniment.

Like the attendant eczema, phlyctenulosis is certainly the result of an endogenous infection, and without doubt—in my mind, at all events—this infection is from the alimentary canal—intestinal intoxication, as it is phrased nowadays. The investigations of Weeks and others, who have usually found the *Staphylococcus aureus* or *albus* present in the phlyctenules, as well as in the eczematous nodules, make it clear that these are the micro-organisms which, having gained entrance to the blood-stream, are commonly responsible for both the skin and the ocular lesions.

How much significance should be attached to the frequent presence of indican in the urine of those suffering with phlyctenular ophthalmia I do not pretend to decide; but investigators who do attach importance to this symptom, and who look for it systematically, report its presence, in large or moderate amounts, in nearly every case of the disease.

If there is any tuberculous affection—or, if you please, pseudotuberculous affection—that responds to local and constitutional treatment in the prompt manner that phlyctenular ophthalmia commonly does. I do not know of its existence. Certainly, true ocular tuberculosis shows no such response, even though tuberculin be employed; on the contrary, it is very intractable and difficult to control.

There are, indeed, few ocular maladies, in my experience, more amenable to judicious therapeutic measures than phlyctenular conjunctivitis and keratitis, and especially is this true of the many cases occurring in childhood. The measures which I employ are regulation of the patient's diet and habits of life, an energetic purgative, which should contain calomel, given at the outset and repeated, perhaps, in forty-eight hours, followed by the administration of syrup of the phosphates of iron, quinin and strychnin—rather than the commonly prescribed syrup of the iodid of iron—and atropin and yellow oxid of mercury ointment as routine local applications. I may add that I feel not a little "disgruntled" if the majority of my patients are not well in considerably less than three weeks.

## CONCLUSIONS

The evidence adduced in support of the doctrine that phlyctenular ophthalmia is a tuberculous or pseudotuberculous lesion is far from convincing.

The frequency with which the subjects of phlyctenulosis give a positive reaction to diagnostic tuberculin tests is of little significance in view of the fact that the same tests show a scarcely smaller percentage of positive reactions in healthy persons.

There is excellent authority for the contention that "until some responsible observer has demonstrated the presence of the tubercle bacillus in an extended series of phlyctenules," or, at least, until it has been shown to be sometimes present, the assertion that the affection is, in any sense, tuberculous is without warrant.

In the present state of our knowledge of the etiology of phlyctenulosis, the employment of tuberculin as a therapeutic agent in this affection is unjustifiable, not only because the ill-considered use of tuberculin is capable of doing much harm, but also because the clinical evidence shows pretty clearly that, if it is not actually harmful, it surely is not helpful. Definite clinical signs of the existence of tuberculosis, apart from the ocular inflammation, may justify the administration of tuberculin, but even in such circumstances the effect on the eye could be only indirect.

As phlyctenulosis is essentially a disease of childhood, and the typical cases occur not in adult life, but in children, it is from the study of these cases that trustworthy conclusions as to the etiology of the affection are to be drawn. The study of these childhood cases shows, from the almost constant association of facial eczema with the ocular inflammation, that phlyctenular ophthalmia, as was formerly very generally held, is an ocular eczema, due, for the most part, like the facial eczema, to intestinal intoxication, and that tuberculosis is seldom, if ever, an etiologic factor.

Cathedral and Howard Streets.

# MEMORANDA

# A STUDY OF THE EFFECT ON HETEROPHORIA OF THE CORRECTION OF AMETROPIA

## WITH A CONSIDERATION OF SOME ASSOCIATED CONDITIONS *

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This analysis was undertaken to determine the effect on heterophoria of the correction of ametropia. In the development of the paper the relation of other factors to the problem brought in additional data. The opportunity which this afforded of analyzing these correlated conditions was embraced and the results incorporated.

The material used was supplied from my private case-records. No case was used unless at least a month had elapsed between the original and final tests. In more than two-thirds of the cases several years intervened, the second determination being made on the patient's return for a change of glasses. Only cases were used in which there was equal and normal, or better than normal, vision in each eye, and in which no actual deviation of the visual axes existed. No case in which prisms had been worn was considered available. Presbyopia was excluded. Only cases were used in which uniform tests had been employed in the various determinations.

As there is a marked discrepancy between some of the results given here and those heretofore published by other writers, it is well to state the methods employed by me in making the various tests. In testing both the far (6 meters) and the near (1/3 meter) status of the muscle balance a colored Maddox rod was used, a light 1 cm. in diameter serving for

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the distant object and a small electric lamp for the near object. Loose prisms or the phorometer was used to measure the degree of imbalance for both the vertical and lateral muscles at infinity and the rotary prism at near. Abduction and adduction were determined by loose prisms. In the past few years when a patient with but slight effort reaches an adducting power of 15 degrees it has been recorded 15 + as experience has shown that these patients readily acquire a high prism adduction by exercise or by knack.

TABLE 1.—MUSCLE BALANCE AT FIRST AND AT FINAL TEST

| | Far | | Near | | | | |
|---|---|---|---|---|---|---|---|
| Ametropia | First Test | Final Test | First Test | Final Test | Hyperph. Per Cent. | Ab. | Ad. |
| H+Ah | Es. 2.5 | Es. 1.75 | Ex. 5.0 | Ex. 4.5 | 26 | 6.0 | 17 |
| H+Ah | Ex. 1.62 | Ex. 1.5 | Ex. 8.0 | Ex. 9.0 | 59 | 6.5 | 14 |
| H+Ah | Orth. 0.0 | Ex. .37 | Ex. 6.5 | Ex. 6.0 | 40 | 6.0 | 17 |
| H+Ah | Es. 2.0 | Es. 2.62 | Es. 6.62 | Es. 3.62 | 25 | 5.5 | 20 |
| H+Ah | Es. 6.0 | Es. 4.62 | Es. 2.5 | Ex. .75 | 57 | 7.0 | 20 |
| Ah | Es. 1.37 | Es. 1.0 | Ex. 4.0 | Ex. 4.5 | 50 | 6.0 | .. |
| Ah | Ex. 2.50 | Ex. 1.87 | Ex. 7.0 | Ex. 4.0 | 50 | ... | .. |
| M+Am | Es. 2.25 | Es. 1.50 | Ex. 1.5 | Ex. 1.25 | 35 | 6.0 | 19 |
| M+Am | Ex. 2.75 | Ex. 3.0 | Ex. 10.0 | Ex. 11.0 | 61 | 7.0 | 10 |
| Amh | Es. 1.75 | Es. 1.62 | Ex. 2.37 | Ex. 4.75 | 20 | 7.0 | 12 |
| Amh | Ex. 1.62 | Ex. .62 | Ex. 13.0 | Ex. 13.0 | 20 | 7.0 | 14 |
| Amh | Orth. 0.0 | Ex. .50 | Ex. 7.0 | Ex. 7.0 | 20 | 7.0 | 15 |
| Anisom | Es. 2.0 | Es. 1.37 | Ex. 6.0 | Ex. 5.62 | 50 | 6.0 | 16 |
| Anisom | Ex. 2.5 | Ex. 1.25 | Ex. 9.0 | Ex. 10.0 | 40 | 7.50 | 12 |

The average hypermetropia in the cases of H+Ah with esophoria for distance and exophoria for near equals 1.50 D.

The average hyperopia in the cases of H+Ah with exophoria for distance and near equals 1.12 D.

The average myopia in the cases of M+Am with esophoria for distance equals 1.87 D.

The average myopia in the cases of M+Am with exophoria for distance equals 2.12 D.

For the purpose of study the cases were divided into groups having like ametropia, and these were subdivided into groups having similar muscle imbalance for distance (Table 2).

TABLE 2.—GROUPS AND SUBGROUPS OF CASES

| Ametropia | No. of Cases | Muscle Imbalance for Distance | Per Cent. |
|---|---|---|---|
| H+Ah | 236 | Orthophoria | 10 |
| | | Esophoria | 62 |
| | | Exophoria | 28 |
| Ah | 20 | Esophoria | 60 |
| | | Exophoria | 40 |
| M+Am | 87 | Esophoria | 60 |
| | | Exophoria | 40 |
| Ahm | 30 | Orthophoria | 24 |
| | | Esophoria | 52 |
| | | Exophoria | 24 |
| Anisometropia | 24 | Orthophoria | 15 |
| | | Exophoria | 25 |
| | | Esophoria | 60 |

There were 145 cases of H + Ah, with esophoria for distance and exophoria for near. The average esophoria for distance at the first test was 2.5°, and at the final test 1.75°. The average exophoria for near at the first test was 5°, and at the final test 4.5°. Average abduction was 6°, adduction 17°. Hyperphoria in 26 per cent.

There were 66 cases of H + Ah, with exophoria for distance and near, greater for near. Average exophoria for distance at the first test, 1.62°, at the final test 1.5°. Average exophoria for near at first test 8°, at the final test 9°. Average abductions 6.5°, adduction 14°. Hyperphoria in 59 per cent.

There were twenty-one cases of H + Ah with orthophoria for distance and exophoria for near. The average exophoria for distance at the final test was 0.37°. Average exophoria for near at the first test 6.5°, at the final test 6°. Average abduction 6°. Average adduction 17°. Hyperphoria in 40 per cent.

There were thirteen cases of H + Ah in which there was esophoria for both distance and near with a greater degree for near. The average esophoria for distance at the first test was 2°, at the final test 2.62°. The average esophoria for near at the first test was 6.62°, at the final test 3.62°. Average abduction 5.5°. Average adduction 20°. Hyperphoria in 25 per cent.

There were ten cases of H + Ah with esophoria for both distance and near with a greater degree for distance. The average esophoria for distance at the first test was 6°, at the final test 4.62°. The average esophoria for near at the first test was 2.50°. This was converted into an average exophoria at the final test of 0.75°. Average abduction 7°. Average adduction 20°. Hyperphoria was present in 57 per cent.

There were twelve cases of Ah with esophoria for distance. The average esophoria for distance at the first test was 1.37° and at the final test 1°. The average exophoria at the first test was 4° and the final 4.5°. Average abduction was 6°. Hyperphoria was present in 50 per cent.

There were eight cases of Ah with exophoria for distance. The average exophoria for distance at the first test was 2.50° and at the final test 1.87°. The average exophoria for near at the first test was 7° and at the final 4°. Hyperphoria present in 50 per cent.

Of eighty-seven cases of M + Am, fifty-six had esophoria for distance and thirty-one had exophoria for distance.

Of the fifty-six cases of M + Am with esophoria for distance the average esophoria at the first test was 2.25° and at the final test 1.50°. The average exophoria for near at the first test was 1.50° and at the final 1.25°. Average abduction 6°. Average adduction 19°. Hyperphoria in 35 per cent.

There were 31 cases of M + Am with exophoria for distance, and greater exophoria for near. The average exophoria at the first test was 2.75°, and at the final test was 3°. The average exophoria for near at the first test was 10°, and at the final test 11°. The average abduction was 7°. Average adduction was 10° Hyperphoria was present in 61 per cent.

There were 30 cases of Ahm. In 16, with esophoria for distance. The average esophoria for distance at the first test was 1.75°, and at the final test 1.62°. The average exophoria for near at the first test was 2.37°, and at the final test 4.75°. The average abduction was 7°. The average adduction 12°. Hyperphoria was present in 20 per cent.

There were 7 cases of Amh with exophoria for distance and near. The average exophoria for distance at the first test was 1.62°, the average at the final test was 62°. The average exophoria for near at the first test was 13°, and at the final test 13°. The average abduction 7°. The average adduction 14°. Hyperphoria was present in 20 per cent.

There were 7 cases of Amh with orthophoria for distance and exophoria for near. The average exophoria at the final test for distance was 0.50°. The average exophoria for near at the first test was 7°, and at the final test 7°. Average abduction was 7°. The average adduction 15°. Hyperphoria was present in 20 per cent.

There were 24 cases of anisometropia, in which the average difference in refraction of the two eyes was 2 D. In 15 there was esophoria for distance. The average at the first test was 2°, and at the final test 1.37°. The average exophoria for near at the first test was 6°, and at the final test 5.62°. The average abduction was 6°. The average adduction was 16°. Hyperphoria was present in 50 per cent.

In 6 cases of Amh there was exophoria for near and far, greater for near. The average exophoria for near at the first test was 2.5°; at the final test 1.25°. The average exophoria for near at the first test was 9°, and at the final test 10°. The average abduction was 7.5°, and adduction 12°. Hyperphoria was present in 40 per cent.

An analysis of these findings with a view of determining the influence of the correction of the ametropia on associated heterophoria shows that: When in H + Ah esophoria existed for far and exophoria for near the esophoria was reduced on the average of 0.75°, or about 33 per cent. of the original amount, whereas the exophoria which was present for near was reduced 0.5°, or 10 per cent. of the original amount, a negligible amount. The esophoria in this status of the muscles has been held by some to be an apparent esophoria, the real difficulty being an insufficient convergence power. The correction of the H + Ah would tend further to reduce the positive portion of convergence, and we might expect to find the exophoria for near, at least, somewhat increased; but it remained practically unchanged. The esophoria, however, was reduced, rather indicating that it arises from the convergence associated with the accommodation required for distinct distance vision.

When exophoria existed for both distance and near with a greater amount for near, practically no change occurred in the distance exophoria, and the exophoria for near was increased 1°, or 12 per cent. of the original amount. Here we have a convergence insufficiency with a beginning divergence excess, and the correction of the H + Ah would tend to increase the exophoria for near by reducing the call on the accommodation. Practically the only cases calling for prism exercise fell within this group. Almost all were relieved of symptoms, and in nearly all of those in which the exophoria was not greater than 2° for distance, it was either overcome or a low esophoria was established; but this did not persist for any great length of time after the prism exercise was discontinued. The relation of prism abduction and adduction was in almost every instance 1:2 (6:13), not very different, however, from those in which prism exercise was not required (6:15).

When orthophoria existed for distance and exophoria for near, an average exophoria of 0.37° developed for distance, and the exophoria for near increased 2°, or 33 per cent. of the original amount.

Here we have practically the same condition and the same result as in H + Ah with exophoria for distance and a greater exophoria for near.

When esophoria existed for both distance and near, but was greater for near, the esophoria for distance decreased on the average 0.62°, or 33 per cent. of the original amount, and the esophoria for near was decreased, on the average, 3°, or 45 per cent. of the original amount.

These are cases of convergence excess in which the esophoria for near is decidedly decreased by correction of the H + Ah.

When esophoria existed for distance and near, but was greater for distance, the esophoria for distance decreased, on the average, 1.37°, or 23 per cent. of the original amount, and the esophoria for near was converted into an exophoria of 0.75°, a change of 133 per cent.

This group represents a convergence excess with a relative divergence insufficiency, the correction of the ametropia markedly decreasing the esophoria for near.

In Ah with esophoria for distance, the correction of the ametropia had no effect on either the distance or near heterophoria.

In Ah with exophoria for distance, the exophoria for both distance and near was appreciably diminished, probably the result of the improvement in vision from the correction of the astigmatism.

When in M + Am esophoria existed for distance and exophoria for near, the esophoria for distance was reduced 0.75°, or 33 per cent. of the original amount, and the exophoria for near was reduced 0.25°, or 16 per cent. of the original amount.

The cases in this group are largely accommodative convergence insufficiency without the later-imposed divergence excess. The decrease in the esophoria for distance was due to the beginning development of a divergence excess, 16 per cent. of the cases showing a change from esophoria to exophoria, and 50 per cent. a decrease in the esophoria for distance. The average exophoria for near was but little influenced, but the cases showing a moderate amount of exophoria for near at the first test nearly all showed an increase in the exophoria.

When in M + Am exophoria existed for distance and for near, the exophoria for distance was reduced 0.25°, or 9 per cent. of the original, and the exophoria for near was increased 1°, or 10 per cent. of the original.

In this group there was a convergence insufficiency and a divergence excess, the elements making for the development, in time, of a divergent strabismus. The average change was so slight, however, as to indicate an arrest of this progress, although in a few cases it had made advances.

When in Amh esophoria existed for distance and exophoria for near, the esophoria remained practically unchanged and the exophoria for near increased 2.37°, or 100 per cent. In this group the slight esophoria for distance which was present may be considered a static condition. The exophoria increased to a marked degree, and this may be due to the accommodative effort occurring in the course of the altering refraction which these cases undergo.

When in Amh, exophoria existed for distance and for near, but was greater for near, the exophoria for distance was reduced 1°, or 60 per cent. of the original, and the exophoria for near remained unchanged.

When orthophoria existed for distance and exophoria for near, exophoria of 0.50°, or 60 per cent. of the original, developed, and the exophoria for near remained unchanged.

These two groups show but little change, probably because these cases showed little change in the refraction in the very few cases in which an opportunity had been presented to re-refract after the lapse of time, and a disturbance of the relation between accommodation and convergence was not induced by the correction of the refraction error. When the few cases were assembled into three separate groups, the number in each was so small that deductions were difficult to make.

When in anisometropia esophoria existed for distance and exophoria for near, the esophoria for distance decreased 0.50°, or 25 per cent. of the original, and the exophoria for near was practically unchanged.

When in anisometropia exophoria existed for near and distance, but greater for near, the exophoria for distance decreased 1.25°, or 50 per cent. of the original, and the exophoria for near increased 1°, or 11 per cent.

These cases showed no greater degrees of heterophoria than the other forms of ametropia, and were little changed by the correction of the error of refraction, except that those showing exophoria for distance had this decreased by equalization of the visual acuity of the two eyes.

In a short series of cases such as was here analyzed, it is possible for a few cases of marked heterophoria to produce an average relatively too high or vice versa. It was thought that this might, in a measure, be a possible source of error, and be neutralized by giving the percentages, in the two main groups, M + Am and H + Ah, of cases showing a similar change. These groups were further analyzed to determine the relation existing between the abducting power and the distance imbalance.

Of the cases of H + Ah with esophoria for distance and exophoria for near, in 25 per cent. there was an increase in the esophoria, the average being 2.37°, and the greatest 4°. In 57 per cent. there was a decrease of esophoria, the average being 2°, the greatest 9°. In this series there was a much larger number of cases showing an appreciable decrease of esophoria than was the case in other of the H series. The average abduction equaled 6°. In 30 per cent. in which it was above the average, the esophoria equaled 2.37°. In 70 per cent. in which it was below the average, the esophoria equaled 2.62 per cent. In a series in which abduction was 5° or below the average, esophoria equaled 3.5°, and in a series in which it equaled 7° or more than the average, esophoria equaled 1.75°.

Of the cases of H + Ah with exophoria for distance and near, in 35 per cent. there was an increase in the exophoria. The average was 1.37°, and 40 per cent. showed a decrease, the greatest being, 2°, the least 1°.

The average abduction was 6.5°. In 50 per cent. it was above the average, and in 40 per cent. it was below the average, but in both the average exophoria equaled 1.5°. In a series in which it was 5° or below, the average exophoria for distance was 3.5°.

Of the cases of M + Am with esophoria for distance, in 30 per cent. there was an increase in the esophoria, the average increase was 1.12°, and the greatest was 3°.

Abduction was below the average in esophoria associated with ametropia of any form, and was of equal degree both in the myopic and hyperopic groups.

Abduction was above the average in exophoria associated with ametropia of any form, and was about equal in exophoria for distance both in the hyperopic and the myopic groups.

Abduction was above the average and of equal degree in Amh, no matter what the distance balance.

In 45 per cent. there was a decrease; the average was 2.25°, the greatest 7°.

From the frequency with which hyperphoria occurred in the foregoing cases, it was evident that right hyperphoria prevailed to a marked degree. As in a tabulation of one hundred cases on another occasion the percentage of right hyperphoria was found to be about 70 per cent., a further series of 200 cases, none of which had been employed in the previous computations, was used. In 105, or 52 per cent., there was orthophoria for the vertical muscles. In 95, or 48 per cent., there was hyperphoria. In 76 per cent. of these it was R. H., and in 24 per cent. L. H. As in making the tests the rod had been invariably placed before the right eye, it was thought that this might in some way be the cause of the preponderance of R. H., but Dr. Halleway found the same preponderance of

R. H. when the rod was placed in front of the left eye. Possibly the fact that the right eye is usually the dominant eye is a factor. Forty-six per cent. of H + Ah, 50 per cent. of Ah, 53 per cent. of M + Am, 40 per cent. of H, 38 per cent. of Amh and 50 per cent. of anisometropia showed hyperphoria.

In 74 cases (78 per cent.) it was less than 1°, in 38 cases (40 per cent.) it was 1.4°, in 9 cases (9.5 per cent.) it was 1°, in 3 cases (3 per cent.) it was 1.5° and in 5 cases (5 per cent.) it was 2°. In 4 cases (4 per cent.) it was over 2°. It was found that in less than 1 per cent. was the hyperphoria appreciably altered by correction of the ametropia.

Hyperphoria was present more often in exophoria for distance than in esophoria for distance in the proportion 46: 36. It was least frequent and occurred in the same ratio (20 per cent.) in all forms of heterophoria associated with Amh.

## CONCLUSIONS

On the whole, it cannot be said that the correction of ametropia greatly influences the associated heterophoria, some of the change here noted being the result of the natural progress of such associated conditions, as evidenced by the fact that a study of a series in which but two weeks intervened between the tests before and after the ametropia had been corrected showed even less change. In a short series of H + Ah with esophoria for distance, exophoria first test 1.37°, final test 1.25°; exophoria for near, first test 6.5°, final test 6.5°.

Considering only the heterophoria, it may be said that the correction of the ametropia reduces the esophoria for distance when associated with H + Ah, and tends to increase the exophoria for near.

When esophoria exists both for distance and near, the heterophoria at both distances will be reduced and the esophoria for near may be converted into an exophoria. Esophoria for distance and likewise an exophoria for near will be reduced by the correction of M + Am.

Exophoria for distance and near will be practically unchanged by the correction of a coexisting M + Am.

Esophoria for distance will be little changed or may go over into an exophoria by the correction of an associated Amh. An exophoria for near, under like conditions, will be increased.

Exophoria for distance in anisometropia will be decreased by the correction of the ametropia.

In no group of cases did the average abduction exceed 7°; the average in all groups was 6.5°.

Hyperphoria occurs in less than 50 per cent. of cases of ametropia. It is somewhat more frequent in H + Ah. In more than three-fourths of the cases it is less than 1°, and in less than 10 per cent. it is 2° or more. Right hyperphoria vastly predominates.

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# MEMORANDA

# SAFE AND SPEEDY EXTRACTION OF THE IMMATURE CATARACTOUS LENS FOLLOWING PRELIMINARY CAPSULOTOMY *

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The contentions embodied in this thesis are:

1. An ample, properly executed preliminary capsulotomy has, in mature cataracts, many advantages over immediate capsulotomy or capsulectomy.
2. In morgagnian cataract it enables one to make what is difficult otherwise, namely, an efficient opening in the capsule.
3. The extraction of the clear lens in high myopia is feasible by this method, and
4. It will render immature cataract operable after a six-hour interval, leading up to a clean extraction with rarely cortical débris left behind.

These contentions are submitted in the foregoing order that their correlated value may be presented and that the subject-title shall receive final and full consideration. It is generally stated by most writers that the lens-capsule is a highly elastic membrane which, on section, curls outward. It is very doubtful if this statement be true; not as to its curling outward, which is thought to be due to another cause, but as to its elasticity. If it were elastic and under tension it would gape only to incisions at right angles to the lines of traction; that a crucial incision should gape would require elastic fibers radially disposed. This is simply a matter of physics. There is nothing to show histologically the presence of elastic tissue in the lens-capsule; on the contrary, it is a homogeneous

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

hyaline structure which does not stain, nor has the most searching microscopic examination shown elastic fibers therein. Neither can the gaping of the capsulotomy wound be explained on the basis of inherent elasticity in the lens fibers themselves. To quote Tscherning, "The contents of the crystalline lens are composed, in the adult, of two parts: the nucleus, which cannot change its form, and the superficial layer which, on the contrary, possesses this faculty to a very high degree; its consistence is very nearly that of a solution of very thick gum." Quoting Hocquard, he continues, "The whole is surrounded by a capsule which is inextensible or very nearly so." In fact, the laws of dynamics and of finely coordinated muscular action elsewhere in the body demand that, for accommodation, the lens capsule shall be inelastic." In an address delivered in the University of Oxford, Sir William Job Collins says:

> The lens is truly described as very elastic in the sense of readily tending to resume its original form after the withdrawal of any force that has modified its form or volume. I should not be disposed to allow that the same elasticity is true of its capsule, though this is alleged by most authors. From a study of it in dissection and in operations, I should be more disposed to say, with Bowman, it is "a hard and dense structure and determines the exact outline of the lens." The capsule is composed of albuminoid substance not of elastic tissue and betrays no trace of cellular structure; on laceration it gapes and sometimes curls in a fashion suggesting that *it is held taut by the reefing lines that bind it to the ciliary processes.* [Italics mine.—H. E. S.]

Underlying the anterior capsule of the lens and forming with it an intimate union is the epithelial layer whose cells by elongation become lens fibers. The well-known reproductive faculty inherent in these cells, and that any epithelial residue left clinging to the capsule may by proliferation produce a secondary membrane, make imperative that as little as possible of this be left in the eye. A close examination of secondary cataracts will disclose a small opening in the capsule, lens matter at the periphery of the pupil and growing out into this new lens fibers in the visual aperture. Incidentally, this is one of the arguments against a capsulectomy. The foregoing, relative to the capsule, is germane to what shall follow when technic is taken up, to the explanation of what changes take place in the interval between capsulotomy and the extraction proper and to the question why, to secure such altered relationship, an ample capsulotomy is necessary. Reference to these will later be made.

A historical review of the operation of primary capsule rupture need not here be made, for Dr. Percy Fridenberg[1] has so exhaustively covered the ground in a paper read before this Section in 1912 that he leaves nothing to be added. I shall, however, avail myself of frequent quotations from this paper to adduce arguments in support of my several contentions.

It is a reasonable assumption that the lens-capsule is more or less adherent to the epithelial layer immediately beneath it; that it is non-elastic; that it is the traction of the reefing lines that causes it to gape when divided, and that, in proportion to the amplitude of such division, it will release itself from the underlying cortex. With no knowledge of the work of earlier investigators I worked out for myself many years ago a method of preliminary capsulotomy which had for its objects a knowledge in advance of the size of the corneal section to be required for an easy exit of the lens and to make certain an ample and sufficient capsulotomy with the maximum of precision and the minimum of risk. At this time the knife-needle of Knapp was being used but, finding with this that the making of the second limb of the crucial incision became next to impossible, because of early loss of aqueous and the obliteration of the anterior chamber, I supplanted it by a miniature scalpel of my own design which obviated this difficulty. A sketch of this is here inserted (Fig. 1).

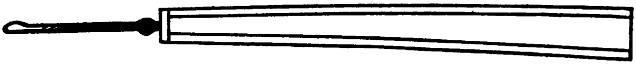

Fig. 1.—The capsulotomy knife.

I have in former papers gone over the matter to follow, but larger experience only makes more certain the truth of the statements therein contained. To restate them: The advantages of a preliminary capsulotomy are many. By the ordinary method some of the difficulties are inherent in the instrument itself, but more and greater ones in the conditions under which it must be used. The straight capsulotome is kept sharp with difficulty. It cuts only when drawn toward the operator; at right angles to this it simply scratches. As soon as the section is made the iris contracts, the lens comes forward and the anterior chamber is abolished. Into this narrow space must be passed a straight instrument between cornea and lens; it is impossible to do this and reach below the anterior pole of the lens without pressing it more or less backward, and at this stage the lens, held only by its suspensory ligament, is mobile and easily dislocated. More than this, the operator in his zeal to make an efficient opening in the capsule is apt to rupture this ligament, and loss of vitreous ensues. There are no means of knowing that the capsulotomy has been inadequately performed until the lens, when pressure is applied to the globe, refuses to come forward. In this event, either the fixation forceps must be reapplied, a difficult and dangerous thing to do on an opened eye, or the cystotome reintroduced without its help, in the trust that the patient will keep still. Narrowing of the pupillary space by the contraction of the iris leaves insufficient room in which to work, and it is a difficult matter not to wound it or entangle it in the tip of the instrument. If the bent cystotome of Knapp is used, these difficulties are enhanced, and not only are there greater obstacles to removing cortical fragments, but a peripheric capsulotomy leaves two thicknesses of capsule in the pupillary space and more often a secondary operation is required. Should blood enter the anterior chamber, the difficulties mentioned before are further increased. None of these difficulties accompanies a preliminary capsulotomy as described in this paper: 1. The knife can be made superlatively sharp, and efficiently cuts the capsule in both directions. 2. Only through the grossest carelessness can the suspensory ligament be ruptured or the lens dislocated, for this is held firmly in place by the vitreous behind and the aqueous in front. 3. Ample space is given through the dilated pupil, and the point of the knife may be kept fully in view during the division of the capsule, and, furthermore, the operator has the satisfaction of knowing that this is adequately performed. 4. The iris cannot be wounded or entangled in the instrument, and there can be no bleeding to obscure the field of operation. In brief, it simplifies and makes certain a step in the operation which is usually fraught with difficulties and dangers. Also when the corneal section comes to be made the iris does not contract so readily, and there is less interference with the extrusion of the lens. It has the further advantage that one can tell in advance of the section just how large this needs to be. If the cut in the capsule is practically invisible, a large nucleus is present. If semigelatinous matter exudes, it is smaller, and if a milky liquid it is smaller still. It is an extremely difficult, if not impossible, task to estimate, with any degree of accuracy, from ophthalmoscopic examination alone, just what is the condition of cortex and nucleus in a cataractous lens, mature or immature. Preliminary capsulotomy reveals the matter and puts us on our guard in some cases. If gelatinous matter exudes we have a soft cortex and probably adherent lens fibers. This is true even if the opacity extends clear up to the anterior capsule and where we would be led to expect, *a priori,* a clean exit of the lens. With an ordinary capsulotomy this means a mushy extraction and much lens matter left in the eye. Even if no operative accident occurs and a nice clear black pupil is secured, the appearance of the eye at the first dressing tells the lamentable tale. Here comes in the advantage of the six-hour interval before the extraction. The action of the aqueous separates the adherent cortical fibers from the capsule, and a clean extraction follows. It is truly marvelous how little pressure is needed to deliver the lens; often it follows the knife on completion of the section. Several operators have written me that this unexpected result was somewhat embarrassing, but it need not be so if it is anticipated. In operating on a morgagnian cataract by the usual method difficulties arise because, on essaying the capsulotomy, the instant the point of the instrument penetrates the capsule the liquid cortex escapes through the puncture, and before this can be extended to an incision, the sac collapses, recedes from the instrument, no further opening is made and there remains in the eye the nucleus in its capsular envelope with an aperture therein too small to admit of its exit. Nothing safely can be done in the way of a more extensive capsular laceration, as the collapsed sac recedes from the point of the cystotome. There is a relaxed zonula, no traction on the capsule, and there remains for further instrumentation only that with the sharp hook or the Daviel spoon. It is not necessary to dwell on the operative difficulties which must then be faced or the dangers to the integrity of the eye. With a preliminary capsulotomy the fluid cortex simply mingles with the aqueous, the sac does not collapse or recede, there is no difficulty in making an ample opening, and at leisure we may observe the size of the nucleus and proportion the extent of the corneal incision for its easy exit. The section may then be made with the angular keratome, and at once if it is so desired. There is no advantage to be gained by waiting in morgagnian cataract, for there is no longer cortex to be dealt with. An operative point is here apropos for such cases and for those in which a primary capsule rupture is to be made instead of the usual capsulotomy. It occasionally happens that sufficient aqueous escapes through the corneal puncture to make the anterior chamber so shallow that section would be difficult. In this event, the tip of the McKeown irrigator is applied to the corneal opening and the anterior chamber filled out with normal salt solution. The operation may then proceed as though this incident had not occurred. It does not need any argument to prove that the smaller the corneal section consistent with an easy exit of the lens, the better are the patient's chances against infection and for an uninterrupted healing of the wound, to say nothing of the larger probability of the surgeon making this in a single plane and with greater facility.

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[1] Fridenberg, Percy: Preparatory Capsulotomy in Extraction of Immature Senile Cataract, The Journal A. M. A., Sept. 21, 1912, p. 1065.

It is conceded that operative difficulties and postoperative dangers attend the removal of a cataractous lens in which there is still much transparent cortex; also, it is certain that a method which would minimize these dangers would be of inestimable value to both surgeon and patient. It is also true that it would be better still if all media could be removed which are opaque or which might become so, provided always that in so doing no greater risks, immediate or remote, are incurred. Given a case in which, from advancing opacity in the lens, there results diminished visual acuity, industrial incapacity and mental distress, several courses are open to us. We may advise, as was formerly the almost universal rule, that the patient wait until complete opacification occurs and the cataract becomes operable. This meant for the sufferer more mental distress, a gradually lessened earning capacity, perhaps even poverty and the loss of all the pleasures of life which are dependent on good vision. To make matters worse, there was the haunting element of uncertainty, for no man could say how long might be the weary waiting, or whether after years of groping in semidarkness, even then operable maturity would result. The second course open would be an extraction after the usual method, with all its attendant dangers, operative and post-operative, and the rather small chance of a final good visual result. The third choice would be artificial induction of operable maturity by Förster's, McKeown's or some other method; the fourth, extraction in the capsule, and the last, safe and speedy extraction by my own method.

It is in the hope that the first shall be last and the last shall be first of these several choices, that this paper is presented for your consideration.

Against the second course the profession is practically united, although it is true that in highly skilled hands, with irrigation to remove cortical débris, with preliminary iridectomy and subsequent needling, a fair proportion of successes may be attained; but no one will deny that the risks are greater, the convalescence more prolonged and the final visual results poorer than would be the case in mature, operable and fully opaque cataract.

The third course is artificial maturation; operations intended for this purpose have not attained success and have fallen into disuse. None of the methods used seem to produce any uniform or decided effect. Puncture of the capsule advised by von Graefe in 1864 and reintroduced by Correnti in 1872 fails for the reason that *punctures* in the capsule do not gape, and there are formed (if any effect is produced) simply insulae of opacity just beneath them and no separation between capsule and cortex. McKeown injected fluid under the lens-capsule. This procedure, though successful, was difficult and dangerous. Does it not seem more rational to bare the lens of its capsule and let the fluids of the eye itself do this effective work?

The fourth course we might follow is extraction in the capsule: against this much must be said. If it could be done with the same proportion of immediate or ultimate successes or with no greater operative risks, this method would be ideal, for it removes in one step every impediment to the clear passage of light into the eye. Unfortunately, the operative risks are appalling and only in hands especially trained under personal tutelage of its one able exponent are these, even then, brought within reasonable comparison of the time-tried classic methods. Barring operative accidents, the immediate results are brilliant; but there are the later ones to be considered. The updrawn pupil (in spite of the large iridectomy) which is later present in these cases points to a dislocation of the vitreous, and I am credibly informed by surgeons of the highest authority in India that there is a slow degenerative change which takes place in these eyes and ultimately leads to much loss of vision. On theoretical grounds the procedure is harsh and unsurgical. To use force sufficient to rupture the zonula, applied from behind (for the resultant force must come from this direction) even if there results no loss of vitreous, is in my opinion a damaging traumatism and a menace to the future integrity of the eye. I have done successful extractions in the capsule, but they were operative mistakes following an insufficient capsulotomy by the usual method. In skilled hands it might possibly be the operation of choice where there was a deep anterior chamber and a tremulous iris, for here one has a weakened zonula; but as a routine measure or for the occasional operator it has my unqualified disapproval. I have had no experience with the manner of separating the lens from its zonular attachment by any of these four methods, namely, suction, traction, lateral pressure or rotation in the patellar fossa; of these the latter appeals to me as more likely to attain the desired result with a minimum of traumatism.

This reviews other methods of dealing with immature cataracts; in my own the capsule is opened by as large a crucial incision as it is possible to make, done as a preliminary measure six hours before the extraction proper. In my own experience, confirmed by that of others, this is sufficient to produce almost invariably a complete separation between capsule and cortex, to make operable cataracts in any stage of maturity leading up to a clean extraction and rarely leaving cortex behind. It has long been my conviction that the extraction of the clear lens in high myopia was feasible by this method, but I had no opportunity to test this personally. In six of the cases analyzed later in this paper it was carried out and all were successful. One could put the procedure to no greater test than this, for surely, if it led up to a facile extraction in these cases, it should not fail if the lens is becoming opaque. No originality is claimed for the preliminary capsulotomy, and whatever merit the method may possess lies in the technic, which is original, and the discovery that immature cataracts may be rendered safely operable in a few hours. If the capsule is inelastic and held taught by the insertion into it of the reefing lines of the zonula, it follows that in proportion as the incision into this is nearer the point of insertion of these lines, the greater will be the retraction; and the more nearly this incision approaches in form an equal-armed cross, the more perfectly will the capsule retract from the lens surface, leaving it open to the imbibition of aqueous. Assuming this to be true, it follows that the technic which will insure this large opening is a matter of importance.

It is a matter of difficulty to convey clearly in words such a description of even the simplest operative procedure that he who reads may run—and do it in like fashion. This has been brought home to me by much correspondence from those to whom my previous descriptions were not clear. To this I have appended illustrations which I trust may help to clarify whatever may be lacking in the written word.

In the first place, the shape of the knife is important if one is to make with it the necessary excursions in the anterior chamber and not get into trouble. The blade length should be 2 mm., the belly rounded, the point obtusely ground and the whole acutely sharp from end to end, for heel, belly and point must successively come into use as the cut progresses.

The pupil having been fully dilated by a 2 per cent. solution of homatropin, the proper preparations of asepsis and cocainization carried out and the speculum inserted, the operator, if for the right eye, takes his place at the head of the patient. Taking a large grasp with the fixation-forceps over the insertion of the internal rectus, with the little and ring fingers resting on the patient's temple, the capsulotomy knife, held as a pen with the cutting edge of the blade downward, is thrust through the middle of the superotemporal quadrant of the cornea, pushed away from the operator through the anterior chamber until the blade reaches the lowest possible point of the dilated pupil and rests on the vertical meridian of the lens; the handle is now raised until the blade penetrates the capsule; the handle is then made to describe an arc of a circle away from the operator, the blade cutting the capsule along the vertical meridian of the lens. To avoid sinking deeply into the lens or producing a dislocation, the shank is gradually withdrawn in describing the arc until at the termination of the cut little more than the blade remains within the eye. The point is now released, the handle brought into its original position, the shank reintroduced and the blade carried over to the margin of the pupil nasalward and onto the horizontal meridian of the lens; the handle is now given a quarter turn toward the operator, thus bringing the cutting surface of the blade against the capsule. Again is the handle made to describe the arc of a circle, this time nasalward, the blade being kept on the horizontal meridian of the lens and the shank gradually withdrawn as the cut approaches completion. The blade is then brought into its original plane of entrance (to avoid enlarging the corneal wound) and withdrawn from the eye. If for the left eye, the operator stands at his patient's left, the corneal entrance is in the tempero-inferior quadrant and the first cut in the capsule begins above on the vertical meridian of the lens, ending below. Otherwise, the technic is the same. The speculum is now removed, a drop of physostigmin (eserin) solution, 1½ grains to the ounce, is dropped into the eye and an antiseptic dressing is applied.

It is evident that on the spherical curve of the lens surface the cut begins with the heel of the blade, is continued with the belly and ends with the point—hence the shape of the blade and the need for cutting sharpness all along its length. That the blade be short is necessary if the proper maneuvers in the anterior chamber are to be made without enlarging the corneal wound, without dislocating the lens, without having loss of aqueous, without cutting too deeply into the lens substance; it is necessary also to success in making the largest possible capsulotomy.

With the ordinary knife-needle, to escape these dangers, only a small crucial capsulotomy is possible. If aqueous is lost while the vertical cut is being made, the transverse one is impossible. If my assumptions of the mechanism of the rapid operability of immature cataracts and the retraction of the capsule be true, then it is essential that the technic here set forth be accurately followed.

Six hours after the capsulotomy the extraction proper is undertaken. I had formerly advised a twenty-four-hour interval, but I fear that this was more from motives of convenience than of necessity. Moreover I could not see that the lens came out any easier in this than in the lesser interval. On two occasions, with the longer waiting, high tension was a complicating factor in the extraction. This has occurred also in the shorter period to other operators, but, while introducing difficulties, has not resulted disastrously, as the extraction has checked the process. Occasionally, some circumcorneal injection was manifest when the dressings were taken off for the second operation. In these cases, to insure proper anesthesia, the cocain was preceded by a drop of 1 : 1,000 epinephrin solution. The extraction proper is done after the usual method, but the operator now knows just how large a corneal section is necessary. If there has been no escape of semigelatinous lens matter from the capsular cut, then the section should comprise the upper two-fifths of the cornea, as the nuclei of immature cataracts are usually large.

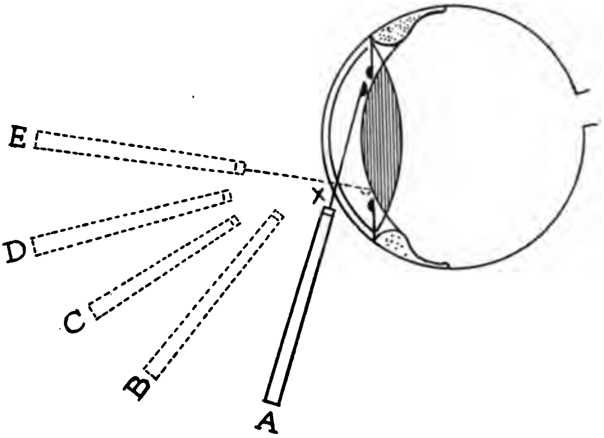

Fig. 2.—Operator standing at patient's head. First plane. Section through right eye on a plane parallel to the long axis of the body. The blade piercing the cornea at *x* is passed down to the lower margin of the dilated pupil and brought successively through positions *b, c, d* and *e,* thereby effecting a long vertical cut in the lens capsule.

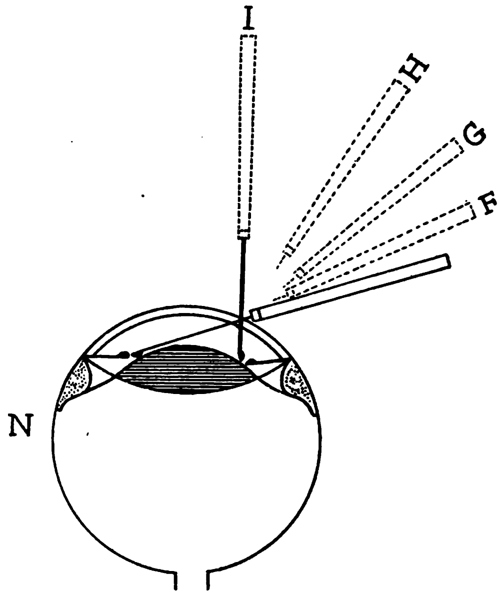

Fig. 3.—Operator standing at patient's head. Second plane. Section through right eye on a plane at right angles to long axis of body. The blade withdrawn from position at e (first plane) is passed to the nasal side (*N*) of the dilated pupil and brought successively through positions *f, g, h* and *i,* thereby effecting a long cut in the lens capsule at right angles to the first incision.

Each operator may elect whether or not he shall make a conjunctival flap. My own preference is for this and a large one. Here, again, is another advantage in preliminary capsulotomy. The flap turned down over the cornea, when an ordinary capsulotomy is next to be done, obscures the operative field. When this has been done, the disposition of the flap is no embarrassment.

The matter of simple versus combined extraction is too complex to admit of review in this place. My own choice is for the former, provided the iris is mobile and dilatable. The cosmetic results are better, and there is no risk of adhesions between cut iris surfaces and tags of capsular membrane. With preliminary capsulotomy there is practically never cortical débris to be reckoned with, and the risk of iris prolapse is minimized by the immediate use of atropin following the toilet of the wound. There are surgeons, however, who prefer the combined extraction as a routine procedure. There is much to be said in its favor, and for the occasional operator who has not the facilities of special hospitals or trained ophthalmic nursing it is perhaps the safer method. That the contentions embodied in this thesis might carry greater weight than would my own unsupported assertions, letters were sent out to members of this Section, whose replies are tabulated here.

RESULTS OF OPERATIONS ON IMMATURE CATARACTS FOLLOWING PRELIMINARY CAPSULOTOMY

| | No. | Per Cent. |
|---|---|---|
| Total number | 180 | .. |
| Of these there were successful | 164 | 91 |
| Total failures | 5 | 3 |
| Partial failures | 11 | 6 |

There were thirty-nine operators who reported cases; of these several reported only one case each. Taking into account unfamiliarity with a new procedure at the hands of many men, and comparing the statistics of results in fully mature and operable cataracts with the percentages here given, in what is admittedly a more difficult and dangerous operation, it will be seen that this method does not suffer by comparison. An analysis of the causes for partial and total failures would make the comparison still better, for of the total failures one had nothing to do with the operation, and the partial failures were tabulated as such irrespective of the final visual results. Of the five total failures, one was due to intra-ocular hemorrhage some days after a successful extraction, with no ascertainable cause; one was due to vitreous prolapse, the nucleus being left in the eye, which was subsequently lost by plastic iridocyclitis; one was due to plastic iritis; the pupil closed in and two cystotomies later failed to keep it open. For the remaining two no cause was given. Of the partial failures eight were due to lens matter left in the eye; in one case the operator failed in technic only, the final visual result being good. No cause was given in two cases. Final results were good in three cases, and not reported in eight. Of operative accidents only three are reported; in two the lens was dislocated, and in the third partially so. There were twelve cases reported of postoperative complications; six of these were iridic inflammations, six of high tension; but while these introduced an embarrassing feature, none resulted disastrously. The intervals between capsulotomy and the extraction varied between six hours and ten days, the largest number being twenty-four hours.

Against the many advantages of a preliminary capsulotomy, irrespective of the maturative process on the partially opaque lens, I advanced the objection that the operation requires more time and trouble, as the technic of asepsis must twice be gone over. Dr. Fridenberg says, "We cannot, however, dismiss all objections in a single sentence unless we close our eyes to the fact that it is the danger of infection even more than the time and trouble of disinfection in a second operation to which we hesitate to expose our patient and which we would not risk unless there was something definite to be gained." I am not aware what is the percentage of infection under modern asepticism in ordinary cataract extractions; but whatever this may be, it cannot be smaller than in the series of 180 cases here detailed, for in this it was zero, there occurring no operative infections at all. This objection therefore *can* be dismissed in a single sentence. Compared with capsulectomy, it is an easier, more effective and a more surgical procedure, not to mention that capsulectomy sacrifices the advantage of knowing in advance how large a section one needs to make. It cannot be combined with capsulectomy if properly performed, nor is there need of this, for there remains no capsule in the pupillary space. In the circular letter sent out criticism was invited. The most serious objection was that high tension followed. This occurred in 3 per cent. of the operations. In such cases it is better not to use a speculum but to employ Angelucci's fixation. Also, the section should end with extreme gentleness, as the lens will almost invariably come out with its completion. The next objection in point of importance was retention of lens matter at the time of extraction. This occurred in less than 5 per cent. of the cases. This, too, compares favorably with the percentage of the same mishap in apparently fully mature lens extractions. It usually can be successfully met by irrigations with McKeown's instrument, using normal salt solution; failing the accomplishment by this, needling should give better vision. There are cases which will always require this, for, quoting Risley, "In this class of cases there is usually some involvement of the posterior capsule as a sequel to the uveal disease which caused the cataract, and secondary capsulotomies are frequently required."

It should not be forgotten that a cataractous eye is a sick eye and perfect visual results the exception and not the rule. Two well-known surgeons have written me that they could not see that the lens comes out any easier or cleaner with a preliminary capsulotomy than without. If this be true, why the many procedures which have been adopted for the artificial maturation of the opacifying lens? Why have surgeons hesitated to operate on immature cataracts? Or why should this paper have been written at all? Many of my colleagues have been so good as to express their commendation. They say:

"It is a valuable addition to our technic, the best since McKeown gave us the irrigator."

"Approve of it very much and favor it especially after having had some experience with the Indian Smith operation."

"Consider your method, if carefully performed, devoid of danger and superior to other methods of hastening the maturity of unripe cataracts."

"The intracapsular operation is still the thing we most desire, but if the capsule of the lens is to be left within the eye, there is no operation which compares with yours."

"Believe this is one of the best, if not the very best, operation for immature cataracts."

"Have found the procedure very satisfactory."

"A safer operation than extraction in the capsule."

"Think it good practice and expect to use it more."

"Believe it to be superior to Förster's."

"I am much impressed with the great value of your procedure. For immature cataract, especially in subjects under 55 years of age, in conjunction with irrigation of the anterior chamber it appeals to me to be unexcelled."

These opinions, selected from many others equally commendatory, are from men eminent in the profession and known to all of you.

In conclusion, I wish again to emphasize the advantages of a preliminary capsulotomy as against capsulectomy or that immediately preceding the delivery of the lens. When properly performed and an interval of six hours allowed to elapse between this and the extraction proper, a separation between capsule and cortex will result and so make immediately operable cataracts in any state of opacification; and the delivery of the lens, mature or immature, is cleaner and easier than by any other method.

# MEMORANDA

# MEMORANDA

# THE INFLUENCE OF HOOKWORM DISEASE ON THE EYES

## A STUDY OF FIFTY-THREE CASES *

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Whenever a new disease is discovered or an old one brought into prominence, the students of the various specialties make determined and laudable efforts to establish a serious connection between the disease in question and their particular specialties. The newly-found (in America) hookworm disease and the students of ophthalmology may well be expected, therefore, to become involved in some sort of investigation. In this particular case, then, let us assume that the incidence of eye lesions in connection with uncinariasis might be of interest and importance in two ways:

1. Is the hookworm infection a causative factor in the existing eye lesion?

2. If it is a causative factor, is the presenting eye lesion sufficiently distinctive or characteristic to be of diagnostic aid in the clinical recognition of uncinariasis?

Before attempting to answer let us briefly consider some salient facts and some of the observations of others appertaining to the questions before us.

### GENERAL CONSIDERATIONS

Uncinariasis, or hookworm disease, belts the earth in a zone about 66 degrees wide, extending from parallel 36 north to parallel 30 south. Practically all countries between these parallels are infected, and these countries contain nearly 60 per cent. of the earth's population.[1] The incidence of the hookworm

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[1] Hookworm Infection as a World Problem, editorial, THE JOURNAL A. M. A., June 22, 1912, p. 1946.

ranges from 50 to 90 per cent. of the population of many of these countries. Evidently it is a world problem. In the rural districts of our Southern states approximately 40 per cent. of the people are infected with this parasite.[2]

Zoologists state that there are two different species of hookworm, the European and the American. As all are agreed that their effects are the same when infecting the human family we may, in this study, disregard the helminthologic distinction. The general pathology and symptomatology of the disease has been well covered in a somewhat extensive literature, which I have carefully considered,[3] but comparatively little has been done in the direction of any special study of ocular conditions in connection therewith.

### EYE LESIONS NOTED IN THE UNITED STATES

Hansell[4] has reported the case of a boy aged 15 infected with hookworm and exhibiting serous and hemorrhagic retinitis with characteristic appearances of retinal anemia.

A. W. Calhoun[5] has reported two cases of uncinariasis exhibiting cataracts in patients aged, respectively, 42 and 14 years.

F. P. Calhoun[6] reports four cases of uncinariasis, three of the patients, aged, respectively, 20, 26 and 22 years, exhibiting cataracts, and the fourth showing recurring spontaneous hemorrhage from the upper conjunctival fornix of the right eye.

Mauldin[7] reports a case of hookworm disease showing "iridocyclitis approximating an interstitial keratitis."

Stiles[8] says that in uncinariasis the pupils of the eyes have a decided tendency to dilatation, the patient may show a blank stare, and that night-blindness is reported in a number of cases. In another place Stiles[9] says:

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[2] Fourth Annual Report, Rockefeller Sanitary Commission for the Eradication of Hookworm Disease, 1913.

[3] The bibliography is well known and easily accessible—especially the Porto Rican studies by Ashford, King and Igaravidez, and the American studies by Stiles.

[4] Hansell: Am. Med., 1901, ii, 412.

[5] Calhoun, A. W.: Ophth. Rec., 1904, xii, No. 7.

[6] Calhoun, F. Phinizy: Eye Complications Caused by Hookworm Disease, The Journal A. M. A., Sept. 21, 1912, p. 1075.

[7] Mauldin: Jour. South Carolina Med. Assn., 1910, vi, 521.

[8] Stiles: Osler's Modern Medicine, i, 589.

> If the patient is directed to stare intently into the observer's eyes, there will be noticed a symptom which it is difficult to describe, but which I have found more constant than almost any other noticed, namely: After a moment, the length of time apparently varying slightly, according to the degree of the disease, the pupils dilate and the patient's eyes assume a dull, blank, almost fish-like or cadaveric stare very similar to that noticed in cases of extreme alcoholic intoxication.

Dock and Bass,[10] after a brief consideration of eye-lesions reported by foreign authors, say:

> We have often noticed that the pupil dilates readily and usually remains wide, even in the presence of considerable light, but this is a common symptom of all severe anemias.

The authors of this book report no original investigations.

So far as I am aware, these are the only published reports of observations in the United States of eye-lesions in hookworm disease. In the spring of 1910 I reported to the Greenville (S. C.) County Medical Society the cases of two girls, aged 10 and 14 years. Both had interstitial keratitis, and both were typical hookworm cases, easily admitting of snapshot diagnosis. This was verified microscopically and the patients were turned over to a colleague for thymol treatment. It is true that the family history, while not positively indicating it, was not such as to be above a suspicion of hereditary lues; but the improvement in the ocular conditions after two or three thymol treatments was little short of astounding. I thought I had made a very interesting and valuable observation, and so I had; but the most interesting part of it came a year later when both patients died of tuberculosis — one of the commonly accepted causes of keratitis parenchymatosa.

### EYE LESIONS NOTED IN FOREIGN COUNTRIES

The European observations on the incidence of eye-lesions in uncinariasis are somewhat fuller, yet even so the literature on the subject is apparently not extensive.

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[9] Stiles: Quoted by Ashford and Igaravidez.

[10] Dock and Bass: Hookworm Disease, St. Louis, 1910.

M. Rampoldi,[11] reporting in 1880, noted that in hookworm patients who complained only of a slight asthenopia, or of the lack of ability to fix objects, he found ophthalmoscopically a whitish retinal reflex, characteristic of edema of this membrane. In some cases visual acuity was diminished. In the case of a woman who died of hydremia he found a true exudative retinitis, which reminded him of Liebreich's cases of splenic leukemia. There were also hemorrhages into the retina, and he found lymphoid elements deposited in great quantity between the retina and the chorioid.

Von Nieden[12] notes in these cases a pale conjunctiva and shining sclera. With the ophthalmoscope he observes marked paleness of the blood-current in the contracted arteries, and tortuous veins; sometimes arterial pulsation, porcelain-colored papillae and, above all, characteristic hemorrhages, like those of pernicious anemia, which lie as isolated spots or broad patches on the periphery of the retina and show a tendency to fatty degeneration. There is, he says, sclerosis and fatty degeneration of the vascular endothelium as in the pernicious anemia of *Bothriocephalus latus*. The subjective symptoms he describes as central disturbance of vision with narrowing of the field, anesthesia of the retina, accommodative and muscular asthenopia, with diplopia and a tendency to nystagmus. In spite of this picture, as outlined, von Nieden expresses the belief that the conditions are not due to anemia, but to a toxin. Out of 3,686 cases examined by him he found three cases of bilateral blindness.

Nuel and Leplat[13] say that miners suffering with hookworm frequently show disturbances of vision consisting of fatigue of the eyes without appreciable lesion; a certain amount of hemeralopia, and frequently nystagmus. These authors examined eighty-one cases and found in five the following changes: One showed retinal hemorrhages; another showed numerous retinal hemorrhages in the right eye, especially in the nerve-fiber layer, and a pale papilla in the left eye, with veins somewhat engorged; two other patients showed infiltration of papilla and retina; while the fifth, an albuminuric, had a neuroretinitis.

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[11] Rampoldi, M.: Cong. périod. internat. d'ophth., 1880, Milan, 1881, vi, 283.

[12] Von Nieden: Ophth. Klin., Stuttgart, 1903, vii, 181; Centralbl. f. Prakt. Augenh., 1903, xxvii, 207.

[13] Nuel and Leplat: Ann. d'Ocul., 1889, lii, 150.

A. Nieden[14] announces that the hemorrhages of the ocular fundus are characteristic. He says that they occur more in the periphery of the retina, and are frequently in groups, point-shaped and close together, but without merging into each other. They frequently follow the course of the vessels, seeming to lie in the vessel walls, and appear rather like stripe-shaped exudates accompanying the vessels to the papillary area. In addition there are extensive blood-exudates in the retinal tissue, represented as large plaques with ray-formed margins. The microscope discovers sclerotic and fatty degeneration of the endothelium of the blood-vessels. In most cases the patient does not know that his eyes are affected, and he is troubled with scotomas only when the macula is the chief location of the blood-spots. More marked are the weakness of accommodation, asthenopia, fatigue of the eyes and periodic slight pareses of intrinsic and extrinsic muscles with diplopia and vertigo. Among miners there are frequently photophobia and hemeralopia. Also there is often a moderate narrowing of the field. The visual disturbances, he thinks, are caused not so much by the retinal changes as by the general symptoms of the disease. (Compare this description with von Nieden's observations abstracted above.)

Fischer[15] notes that the hookworm itself has never been found in the eye. Birmer asserted that retinal hemorrhage was pathognomonic of the anemia named after him, but this is incorrect, since it is well known, as Fischer observes, that retinal hemorrhages occur in all forms of anemia, but especially, he thinks, in ankylostomiasis.

Lutz[16] remarks that in ankylostoma infection the vessels of the fundus, especially the arteries, are contracted and the papilla is paler than normal. Lutz has also noted, as a rare sign, chemosis of the bulbopalpebral fold.

R. Rampoldi[17] relates a case of bilateral convergent strabismus, coming on suddenly, and accompanied by homonymous diplopia. Treatment for hookworm was administered and the strabismus disappeared.

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[14] Nieden, A.: Wiener med. Presse, 1897, xxxviii, 1094.

[15] Fischer: Ber. d. ophthal. Gesellsch., Stuttgart, 1892, xxii, 26; Deutsch. med. Wchnschr., xix, 311.

[16] Lutz: Samml. klin. Vortr., Leipsic, 1880-1886, Inn. Med., No. 88.

[17] Rampoldi, R.: Ann. di Ottal, Pavia, 1888-1889, xvii, 170.

Ashford and Igaravidez[18] state that in a series of cases of this disease, 69.5 per cent. had more or less serious disturbances of the eyes. (There seems to be an error in the figures, however, and the percentage should have been put at 34.9.) They go on to say:

> We cannot doubt that much of this was due simply to the degree of anemia, but there remains a considerable percentage . . . with more or less marked visual affections with very little anemia.

They quote Loebker and Bruns as remarking on the pallor of the eye-grounds, pulsation of veins, lowered intra-ocular pressure, venous tortuosity and, commonly, retinal hemorrhages. They note that many authors speak of edema of the papilla. They quote Siccardi as observing nystagmus, diplopia, amblyopia, muscular and accommodative asthenopia and restriction of the field. This author admits the possibility of anemia being the cause of most of the eye symptoms, and that functional visual disturbances may be due to reflex irritation from the intestinal tract, but avers that when we have a retinitis coincident with optic atrophy, the question of the presence of a circulating toxin must be considered. Ashford and Igaravidez emphasize the occurrence of "blurred vision" and night-blindness, and mention the fact that corneal ulcers have been reported, they having seen one case of the latter. They report that Lippitt, with a large experience, states that fully three-fourths of his cataract patients in Porto Rico were anemic.[19]

## COMPARATIVE CONSIDERATIONS

Here, then, we have an array of symptoms and pathologic conditions, any of which — with the exception of cataract, noted and discussed below — might be fully expected to appear in the ordinary run of cases of anemia. The lesion most frequently noted and dilated on appears to be retinal hemorrhage, and one or two of the authors referred to above seem to think that the appearance of these hemorrhages in the peripheral area of the retina is more or less characteristic. This is assuredly a mistake, for such lesions commonly occur in other forms of anemia. Some idea of the frequency of retinal hemorrhages in anemia may be had from the figures quoted by Weeks,[20] which recite that of 238 American cases of pernicious anemia, 31 per cent. had retinal hemorrhages, and of 326 foreign cases 72 per cent. exhibited this eye lesion.

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[18] Ashford and Igaravidez: Uncinariasis in Porto Rico, U. S. Senate Document 808, 1911, p. 96.

[19] Two Japanese authors have had something to say on this subject, but I was unable to find a translator. The references are: Inoshita, Chingai Iji Shinpo, Tokio, p. 1160. Onishi, idem., xiv, No. 4, p. 2. Hisaki, Chiba Igahuhwai-Zassh, Tokio, 1900, No. 47, p. 1. For these and some other references I am indebted to Dr. Charles Wardell Stiles.

In uncinariasis there is a decrease of the hemoglobin content and of the red blood-corpuscles, and a comparative increase in the leukocyte count. When Weeks wrote his book, which was published in 1910, he had probably never given a thought to hookworm disease as having any influence on the eyes, nor, I believe, was there any reason why he should have, but the following brief extracts from his discussion of leukocythemic retinitis and the retinitis of pernicious anemia (which he remarks are very similar) make an interesting comparison with Nieden's report abstracted above: In the first-mentioned form of retinitis he says, "The fundus of the eye usually appears lighter in color than normal . . . the arteries are pale, often somewhat reduced in caliber . . . the veins are dilated, broad and tortuous . . . the retina often becomes hazy . . . numerous hemorrhages occur near the periphery of the retina . . . many striped or flame-shaped hemorrhages may occur in the vicinity of the disk," and in the retinitis of pernicious anemia he says, "The nerve-fiber layer of the retina is often edematous . . . in places the fibers are pushed apart by hemorrhages . . . the elements of the deeper layers of the retina are also separated by the constituents of the blood that have escaped from the blood-vessels . . . the escaped blood-corpuscles undergo degenerative changes . . . white corpuscles are found in the clots, and, in addition, small hyaline masses are present." Indeed, in this brief description one can almost see before his mind's eye one of Nieden's "characteristic" hookworm cases.

In reference to the blurring of vision and blindness mentioned as accompanying uncinariasis, it is to be noted that Kurtsinger[21] collected 198 cases of more or less complete blindness due to hemorrhage from remote parts of the body, the bleeding having been either sudden and profuse — a single hemorrhage — or slow and continuous (this is the method of extraction by the hookworm). It is interesting to observe that out of 189 of these cases, seventy-two were from hemorrhage from the stomach and intestine, and that three followed the application of leeches.

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[20] Weeks: Diseases of the Eye, 1910, p. 766.

Some of the authors quoted above maintain that the presence of the eye-lesions which they have noted must be explained on the theory that there is a specific toxin in hookworm disease. But why should a mere theory be mooted to explain something that is already easily comprehensible and that has been demonstrated time and time again to be based on a well-recognized pathologic principle? Furthermore, if the eye-lesions were caused by a toxin, then those having the heaviest infections should produce the most eye symptoms; but this is not at all the case. It is well to remember that the retinal lesions of anemia are due either to a reduction in the amount of circulating blood, or to a change in the quality of the blood. The hookworm accomplishes both ends, whether or not it is the manufacturer and disseminator of a specific toxin, for it extracts blood in volume, and decreases the hemoglobin and red corpuscle contents. The hookworm, then, causes an anemia, by no means necessarily a toxemia, and any form of anemia may cause the eye symptoms we are discussing — a fact which has been recognized certainly ever since Helmholtz gave us the first ophthalmoscope more than sixty years ago.

In addition to the symptoms mentioned, pallor of the disk, exudative retinitis, retinal edema and vascular tortuosities are more or less common fundus changes in anemia, and cannot be considered as indicating any specific causation.

Dilatation of the pupil is of such common occurrence (a weakening of the iris constrictor) in the various anemias that it is familiar even to the general practitioner; yet many of such cases may be more fancied than real, since there is no fixed physiologic size for the pupil. In the fifty-three cases which I have tabulated I did not once see a really noticeably dilated pupil; but there were very few severe cases in this series. I failed entirely to elicit Stiles' "fish-like-stare" sign, though I made a number of efforts to do so. Ashford and Igaravidez, with their unlimited field of observation, were unable to recognize it.

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[21] Kurtsinger: Quoted by Weeks: Diseases of the Eye, 1910, p. 434.

Imbalance of the ocular muscles and accommodative weakness are not infrequently seen in anemia. It would be curious, indeed, if, in the general myasthenia, the delicate musculature of the eye should escape involvement.

It is hardly fair to accuse the hookworm in cases of hemeralopia and nystagmus. The authors noting these are foreigners, many of whose cases are in miners, who are, of course, as a class, peculiarly subject to these ocular symptoms.

As to nyctalopia, this is a phenomenon known to exist most frequently in tropical and subtropical countries, and it is therefore easy to understand why the principal references to this symptom come from Ashford and Igaravidez in Porto Rico.

F. P. Calhoun[6] believes and Hansell[22] concurs, that the cataracts which he has observed in hookworm patients were caused by a toxin originating from the parasite, and directs attention to the theory that some forms of cataract may be caused by circulating toxins of various sorts — chemical or biologic. It may well be that the hookworm evolves a toxin; Ashford and Igaravidez and others believe so, as I have already remarked, but it has never been demonstrated; and it is certainly strange, if there is such a toxin and it is a cause of cataract, that only five such cases have been reported in our Southern states, where it has been demonstrated that 40 per cent. of the population of the rural districts are the victims of uncinariasis.

Lippitt,[23] with a large experience, states that fully three-fourths of his cataract patients in Porto Rico were anemic. It is well known that cataract occurs more often among the lower classes and the poorly nourished than among persons in better environments. When, therefore, we consider that 80 per cent. of Porto Rico's population is infected with hook-worm,[24] and are consequently in poor hygienic condition, it is

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[22] Hansell: Discussion on article by Calhoun, Footnote 6.

[23] Lippitt: Quoted by Ashford and Igaravidez.

[24] Ashford and Igaravidez: Uncinariasis in Porto Rico, 1911, p. 179.

## EYE-SYMPTOMS IN FIFTY-THREE HOOKWORM PATIENTS*

| Case No. | Severity of Infection | General Condition | Vision Without Correction R. | Vision Without Correction L. | Conjunctiva | Cornea | Anterior Chamber | Retinoscope Refractive Error | Ophthalmoscopic Findings | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Normal | Comp.hy.astig. | Tortuous vessels. Tiny old hemorrhage and pigment left. | |
| 2 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Normal | Hy. astig. | Normal | |
| 3 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Normal | Comp.hy.astig. | Normal | |
| 4 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Normal | Hy. | Normal | |
| 5 | Light | Fair | 15/20 | 15/20 | Folliculosis | Normal | Normal | Comp.hy.astig. | Normal | |
| 6 | Light | Fair | 15/15 | 15/15 | Blepharitis | Normal | Shallow | Hy. | Slight retinal congestion | |
| 7 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Normal | Comp.hy.astig. | Pale retinas, large veins | |
| 8 | Medium | Poor | 15/15 | 15/15 | Pale | Normal | Normal | Emmet. | Pale disks, tortuous vessels | |
| 9 | Medium | Poor | 15/30 | 15/30 | Normal | Normal | Normal | Mixed Astig. | Red disks, tiny hem. and pigment left | Epicanthus |
| 10 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Normal | Emmet. | Retinas congested, disks indistinct | |
| 11 | Light | Fair | Illiterate | | Normal | Normal | Shallow | Hy. | Normal | |
| 12 | Light | Fair | 15/15 | 15/40 | Normal | Normal | Shallow | Hy. | Venous congestion, tortuosities | |
| 13 | Light | Fair | 15/30 | 15/30 | Normal | Normal | Shallow | Hy. | Pale fundi | |
| 14 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Shallow | Hy. | Normal | |
| 15 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | Hy. | Normal | |
| 16 | Light | Fair | 15/30 | 15/30 | Normal | Normal | Shallow | Hy. | Normal | |
| 17 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Normal | Emmet. | Central veins full | |
| 18 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Normal | Hy. | Veins full and tortuous, disks obscure | |
| 19 | Light | Fair | 15/30 | 15/40 | Normal | Normal | Shallow | Hy. | Normal | |
| 20 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Shallow | Hy. | Normal | Epicanthus |
| 21 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | Comp.hy.astig. | Tortuous vessels | |
| 22 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Normal | Comp. My. astig. | Deeply pigmented fundi, tortuous vessels | |
| 23 | Light | Fair | Illiterate | | Normal | Normal | Normal | Hy. | Normal | |
| 24 | Medium | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | Hy. | Normal | |
| 25 | Medium | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | Emmet. | Normal | |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 26 | Medium | Fair | 7/70 | 6/70 | Normal | Normal | Normal | Hy. | Deeply pigmented fundus, tortuous vessels | Alternating internal strabismus |
| 27 | Severe | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | Emmet. | Retinal congestion, disks indistinct, tortuous vessels | Thymol treatment 4 weeks ago. Numerous ova now in feces. |
| 28 | Severe | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | Emmet. | Right fundus normal, left congested | |
| 29 | Light | Fair | Illiterate | | Normal | Normal | Normal | Emmet. | Pale fundi | |
| 30 | Light | Good | 15/15 | 15/15 | Normal | Normal | Shallow | Comp.hy.astig. | Pale disks | |
| 31 | Light | Good | 15/20 | 15/20 | Normal | Normal | Shallow | Hy. | Normal | |
| 32 | Light | Fair | 15/20 | 15/15 | Normal | Normal | Shallow | My. | Vessels full and tortuous | |
| 33 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Normal | Hy. | Normal | |
| 34 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Normal | Hy. | Pale fundi | |
| 35 | Light | Fair | 15/20 | 15/20 | Normal | Normal | Normal | Hy. Astig. | Vessels full and tortuous | |
| 36 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | .......... | ............... | |
| 37 | Severe | Poor | 15/15 | 15/15 | Normal | Normal | Very shallow | Hy. | Pale fundi | |
| 38 | Medium | Poor | 15/20 | 15/20 | Normal | Normal | Shallow | Emmet. | Pale disks | |
| 39 | Medium | Poor | 15/20 | 15/30 | Normal | Normal | Shallow | Hy. | Fundi pale, disks hazy | |
| 40 | Medium | Poor | 15/15 | 15/15 | Folliculosis | Normal | Normal | Hy. | Pale fundi | |
| 41 | Light | Good | 15/15 | 15/15 | Normal | Normal | Normal | Hy. | Disks pale, vessels tortuous | |
| 42 | Light | Good | 15/15 | 15/15 | Folliculosis | Normal | Normal | Hy. | Normal | |
| 43 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Normal | My. | Normal | |
| 44 | Light | Fair | 15/70 | 15/70 | Chr. congest'n | Normal | Normal | Comp.hy.astig. | Normal | |
| 45 | Light | Fair | 15/70 | 15/70 | Chr. congest'n | Normal | Normal | Mixed astig. | Normal | |
| 46 | Severe | Poor | 15/15 | 15/15 | Normal | Normal | Shallow | Emmet. | Fundi pale, vessels tortuous | |
| 47 | Light | Good | 15/15 | 15/15 | Normal | Normal | Normal | Hy. | Normal | |
| 48 | Light | Poor | 15/15 | 15/15 | Normal | Normal | Shallow | Hy. astig. | Normal | |
| 49 | Light | Fair | 15/15 | 15/15 | Normal | Normal | Shallow | Hy. | Normal | |
| 50 | Light | Good | 15/30 | 15/20 | Folliculosis | Normal | Shallow | Hy. | Normal | |
| 51 | Light | Fair | 15/15 | 15/15 | Normal | Smoky | Shallow | Hy. astig. | Normal | |
| 52 | Severe | Very poor | .... | ..... | Congested | Normal | Normal | .......... | ............... | Interstitial keratitis. Died tuberculosis one year later. |
| 53 | Severe | Very poor | .... | ..... | Congested | Smoky | Normal | .......... | ............... | Interstitial keratitis. Died tuberculosis one year later. |

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* In all the patients the pupils were normal in size and reaction, and the lenses were normal.

certainly not remarkable that three-fourths of all cataract patients in that island are observed to be anemic. Indeed, it is rather more to be wondered at, in the light of these facts, that the percentage of anemic cases in such a series does not go to a much higher figure.

## A TABULATED SERIES OF FIFTY-THREE CASES

Other eye-lesions associated with anemia are some forms of choroiditis, vitreous opacities and palpebral folliculosis. Curiously enough, none of these have been mentioned heretofore in connection with hookworm anemia, yet they must have been present in some of the thousands of cases that have been examined. Doubtless, choroiditis has been overlooked in the frequent retinal involvement, though I saw traces of it in two cases (4 per cent.) of my series tabulated herewith (Cases 1 and 9). Vitreous opacities might, of course, be easily overlooked unless especially sought for. In my series I noted four cases (7.5 per cent.) of folliculosis (Cases 5, 40, 42 and 50).

In the fifty-three cases of my series there were thirty-five males and eighteen females. The ages ranged from 9 to 22 years. The diagnosis of hookworm was made microscopically in every case. Only one patient (Case 27) had been treated previously. Homatropin mydriasis was used in every case for retinoscopy and ophthalmoscopy. It was my impression (without actual timing) that the patients on the whole responded to the mydriatic more quickly than normal persons would have. The refractive errors were found to follow closely the well-known average of persons of like age. The two cases of interstitial keratitis noted have been referred to in the foregoing text. There were no marked eye-symptoms voluntarily referred to by a single patient. In twenty-six of these cases (approximately 50 per cent.) I thought I observed a shallowness of the anterior chamber, one of them (Case 37, a severe case) being especially marked. I cannot insist on this observation, however, since I had no means of accurate measurement, so that the report on this point is but a matter of opinion without substantiation, and I do not, myself, attach any great significance to it. The most common fundus changes were pallor and tortuous vessels, the latter occurring in twelve cases (22.6 per cent.). Retinal hemorrhage had occurred in only two cases (Cases 1 and 9), and then in but slight degree.

### CONCLUSIONS

1. The answer to the first question in the opening sentences of this paper is that hookworm disease, by virtue of the qualitative and quantitative anemia and the general systemic ill condition for which it is responsible, is, in this indirect way, and in this way only, a causative factor in the various eye-lesions which have been described as accompanying it.

2. To the second question the answer is that none of the eye-lesions occurring in this disease are in any sense sufficiently distinctive or characteristic to be of diagnostic value.

The evidence, collected from various sources, seems complete enough, and in addition if, in a systematic examination of the eyes of fifty-three consecutive and unselected cases, we find nothing to point us to a specific causative factor for the eye-lesions, and nothing in the clinical ocular pathology that could be regarded as a distinctive diagnostic aid in the recognition of the systemic disease, we may safely conclude that, in the light of our present knowledge, the association of eye-symptoms and uncinariasis must be regarded as purely incidental, or at least that the eye-symptoms occur only as the sequelae of general pathologic conditions, the principles of whose existence have long been recognized.

It need hardly be pointed out that the importance of these conclusions lies, of course, in the fact that many cases of anemia, with or without eye manifestations, occur without an accompanying hookworm infection, and also that many cases of uncinariasis, with or without eye complications, are encountered which are lacking the symptom of anemia. The latter condition is seen in many apparently robust and even athletic young persons from the rural districts and is especially marked in the negro, who seems more or less immune to the hookworm syndrome, though Burdell[25] has shown that in certain localities in our South 65 per cent. of all negroes are infected with the parasite, and, while acting as intermediary hosts and active carriers of the disease, present no clinical symptoms whatever.

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[25] Burdell: Jour. South Carolina Med. Assn., 1910, vi, 326.

Obviously, it is well to know, when we see the signs of anemia in the eye-grounds, or in the facies for that matter, that we might properly suspect the hookworm of being a possible cause of the anemia; but beyond this our studies of the coincidental eye lesions in uncinariasis have not yet carried us.

# MEMORANDA

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# CONVERGENCE INSUFFICIENCY *

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Convergence insufficiency is a frequent anomaly. Probably 8 per cent. of our eye patients show some degree of it, and this proportion would certainly be increased if every patient that presented himself were carefully tested. This statement by no means implies that in all or even a majority of these cases the convergence insufficiency is responsible for the symptoms. On the contrary, in a great many instances convergence insufficiency — especially the accommodative form — seems to cause no trouble whatever. Still it does cause trouble often enough to make it important for us to consider carefully its causation, varieties and treatment.

In considering a condition like this it is obviously essential that first of all we should have clear ideas of its nature. We may start, therefore, with a definition and say that convergence insufficiency is a condition the distinctive feature of which is that the eyes converge imperfectly or with difficulty, the disability not being due to impairment in power of the internal recti *per se*. It is, in other words, an anomaly of a coordinate movement of the eyes and not of the eye muscles, and as such it is in all ordinary cases a strictly binocular affection, each eye being equally involved.

## DIAGNOSIS OF PURE CONVERGENCE INSUFFICIENCY

A pure case of convergence insufficiency presents the following features:

1. Deviation for distance little or none.
2. Prism divergence (abduction) not excessive.

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3. Prism convergence (adduction) subnormal and difficult.

4. Exophoria for near points marked and increasing with approximation of test-object.

5. Convergence near-point abnormally remote.

6. Lateral movements of each eye inward and outward normal in extent and comitant.

7. Diplopia, if present, not increasing to right or left.

8. Convergence reaction of pupil deficient or absent.

These signs deserve a brief consideration.

1. *Deviation for Distance.* — In pure convergence insufficiency there is rarely more than a centrad of exophoria for distance. Not infrequently there is orthophoria, and occasionally even a centrad or so of esophoria. Even when the convergence insufficiency is beginning to be complicated by a divergence excess so that there is more or less exophoria for distance, this is moderate in amount and always much less than the exophoria in near vision.

The tests for distance should be made both with and without the patient's distance-glasses in order to determine the effect that the latter have on the deviation.

2. *Prism Divergence.*—The prism divergence (so-called abduction) for 5 or 6 meters ranges in general from 3 to 8 (in most cases from 4 to 6) centrads. It corresponds, therefore, to the normal. In some cases it may even be subnormal. When the prism divergence is 9 centrads or over, the condition is no longer a pure convergence insufficiency, but is complicated with a divergence excess. There is then usually a more or less considerable degree of exophoria for distance.

3. *Prism Convergence.* — The prism convergence (so-called adduction) for 5 or 6 meters is often low (varying from 6 to 12 or 14 centrads). Low prism convergence in itself means nothing abnormal, since many persons with normal convergence are unable at the start to overcome prisms, base out, of more than 10 or 12 centrads. In such persons, however, it is evident that this inability is due to lack of facility rather than lack of power, and when they once learn, which they do after two or three trials, they go steadily

and easily upward, so as ultimately to overcome prisms of 30, 40 and 50 centrads. Moreover, when they can overcome a prism at all, they can hold the images together without difficulty. In convergence insufficiency, on the other hand, it often happens, particularly in the serious and troublesome cases, that the patient who can overcome, say, a 12-centrad prism, can only with difficulty and strain pass from this to a 14-centrad; and even when he unites the images with the higher prism, he cannot hold them together for more than a moment. In extreme cases of this sort the inability to overcome prisms, base out, is very marked. I remember one instance in which the patient after weeks of faithful trial was unable to advance beyond a 6- or 7-centrad prism. So in cases of convergence paresis (see below) the prism convergence is extremely low or absolutely nil; and it also, of course, is abolished in complex cases in which a well-marked divergent squint has developed; but the latter are never pure cases of convergence insufficiency.

In most cases in which the prism convergence is low and difficult to acquire, it can still be brought to the normal by persistent training, and it is just in such cases, I believe, that training of this sort, when ultimately successful, is most beneficial.

4. *Exophoria for Near Vision.*—The exophoria for 25 cm. ranges from 6 to 30 centrads. Measurements made with the screen, parallax and phorometer usually agree and are pretty constant, provided the test-object is sharply defined (a fine dot is best for the purpose) and provided the patient is directed to fix and focus sharply on it. If these precautions are neglected, the amount of exophoria may vary widely even during the same examination. It often also varies from day to day, the amount depending on the patient's condition and the energy he is able to exert at the time. It will also be modified by the visual acuity and by the strength of the glasses used, and the effect of the latter should be particularly studied, in order to determine how far the deviation is accommodative in origin. In presbyopes a test should always be made with the reading-glass, and the strength of the latter should always be noted.

The exophoria regularly increases as the test-object is brought nearer the eyes. In convergence paresis this increase is particularly marked and uniform. In fact, in order to make the diagnosis of paresis, we have to make our near tests both beyond and within the usual 25-cm. distance, for instance, also at 1 meter, 50 cm. and 10 cm. (see below, in the discussion of convergence paresis).

However great the exophoria for near vision, in pure cases of convergence insufficiency it is habitually overcome at the ordinary reading-distance, provided both eyes can see the object of fixation. The patient, that is, has ordinarily at near points an exophoria and not a squint, although he may, and often does have momentary failure of binocular fixation with consequent double vision and confusion. It is only when the insufficiency is complicated with other factors (consecutive divergence excess, secondary weakening of the interni) that a constant squint for near points develops.[1]

5. *Convergence Near-Point.* — The convergence near-point (Pc) is abnormally remote. The normal position of the convergence near-point, as determined by a number of measurements, is in adults from 5 to 9 cm.,[2] and in children 4.5 to 8 cm.[3] from the base-line connecting the centers of the two eyes. It can be quite precisely measured as follows: The patient's glasses, or in lieu of these a trial-frame or spectacle-frame, is placed on the patient's nose and is used as a reference plane which is adjusted so as to be just 11.5 mm.[4] in front of the corneal apex when the eyes are directed straight ahead. The apex of a millimeter rule is placed against the point at which the reference plane crosses the nose, and the rule itself is held perpendicular to the plane, so as to lie strictly in the midline. Along this rule the test-object, which consists of a pin with a white head 2 mm. in diameter, or a minute dot on a card, is carried straight toward the patient's nose, the patient all the time being exhorted to converge on it as sharply as he can. The

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[1] Even when complicated with a marked divergence excess, so that the deviation is as high as 50 centrads, the patient may at times get binocular single vision. But in such cases the original condition is always probably a divergence excess, the convergence insufficiency being secondary and of recent development.

[2] Indicating an actual convergence of from 38 to 62 degrees.

[3] Indicating an actual convergence of from 45 to 65 degrees.

[4] The distance is 10.5 mm. if the eye is quite myopic, 12.5 mm. if the eye is highly hyperopic.

moment the object doubles insuperably, or the moment one of the patient's eyes is seen to diverge, the distance of the object from the reference plane is measured. If to this distance 2.5 cm. be added, the result will be very nearly the true distance of the convergence near-point from the intercentral base-line. If the latter distance (PcB) is beyond 8 cm. it denotes some weakness of convergence, and if beyond 10 cm. a well-marked insufficiency. Or we may say that the patient has a convergence insufficiency if his *convergence index*[5] falls below 30.

It is understood that in making this test the patient should try as hard as he can to converge on the test-object and to fuse the images as soon as the object tends to double.

In simple convergence insufficiency the convergence near-point is *absolute,* that is, the patient brings the eyes together so that both are directed at the test-object. When there is exotropia, only one eye is directed at the object and converges on it as it is approximated. In that case the other eye may make no attempt to converge at all, so that as fast as its fellow eye comes in, it goes out. Or, it also may make at least an attempt to converge and, although never actually directed at the test-object, may follow it in its movement toward the eye down to a certain point (*relative* convergence near-point) and then suddenly give up the effort and diverge.

The closer the relative near-point of convergence is, the greater we judge the patient's converging power to be, and the more we can rely on it in supplementing the effect of exercise or of operation.

When the patient has a considerable vertical deviation, his converging power may appear to be poor, when really it is quite good, for when the test-object is brought toward the eyes, he sees two images which

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[5] The convergence index (Ci) $= \frac{100 \times \frac{1}{2} \text{ interpupillary distance}}{\text{PcB}}$

Ci ordinarily ranges from 35 to 50, although it sometimes equals 55 and may even (especially in children) run up to 60 or 63. When Ci is between 15 and 59, that is, for all values commonly encountered in both normal and abnormal cases, the relation holds that C=Ci + 3°, where C is the corresponding convergence of the eyes in degrees. For higher values of Ci the following relations obtain:

| Ci | C | Ci | C |
|---|---|---|---|
| 60-63 | Ci + 2° | 75-77 | Ci — 2° |
| 64-67 | Ci + 1° | 78-80 | Ci — 3° |
| 68-71 | Ci | 81-83 | Ci — 4° |
| 72-74 | Ci — 1° | 84-85 | Ci — 5° |

he cannot bring together because they are on different levels. As he cannot unite them he makes no attempt to converge. In that case the dot used as a test-object should be replaced by a vertical line. The patient will converge on this, because when he does so the two images will unite, even if on different levels, since the bottom of one will join with the top of the other. On such an object a patient will be able to converge sharply, even when for the dot he apparently had very little converging power.

6. *Lateral Movements of the Eyes.*—In a pure convergence insufficiency, the inward and outward rotations of each eye, when moving parallel with its fellow, are normal in extent and comitant. This is found both in monocular and in binocular testing. An eye, for example, which, in attempting to converge with its fellow, halts when its corneal margin is 2 or 3 mm. from the caruncle, will, when tested with the perimeter or tropometer, rotate 45 or 50 degrees inward, and then, following the other eye as the latter moves outward, will turn in fully to the caruncle. Moreover, the movements are comitant, one eye moving as fast as the other without wavering or lagging on the one hand, or overshooting, on the other.

7. *Diplopia.*—Diplopia if present does not increase as the eyes are turned to the right or left. Owing to the natural tendency of the eyes to diverge when raised and to converge when lowered, the crossed diplopia caused by a convergence insufficiency tends to increase in looking up and to diminish in looking down. In slight cases of convergence insufficiency it may be present only in the upper field. Owing to the fact that the diplopia is caused by the inability of the eyes to converge properly, it naturally increases as more and more call is made on the converging power, that is, as the test-object is brought nearer the eyes. In a convergence paresis, this increase is particularly striking and takes place with almost mathematical regularity from distance to near, beginning to be observable at 20 feet or more from the eyes.

8. *Pupillary Signs.*—In cases of marked convergence insufficiency with remote convergence near-point, and particularly in cases of convergence paresis, the convergence reaction of the pupil is diminished or absent. In ordinary cases if we bring an object toward

the eyes and urge the patient to fix sharply on it, the pupils contract as the eyes converge. The moment he gives up the effort of convergence and the one eye flies out, the pupils dilate. The more remote the convergence near-point, the sooner this will occur and the less marked will be the initial contraction of the pupil. In great weakness or in actual paresis of convergence there may be no initial contraction of the pupils at all. In this case if the reaction to light is present we have the inverse of the Argyll Robertson pupil.

### DIFFERENTIATION FROM OTHER FORMS OF EXOPHORIA

The two chief conditions from which a convergence insufficiency is to be differentiated are divergence excess and insufficiency of an adductor.

*Divergence Excess.*—A considerable number of cases of exophoria, probably a third, in fact, of all the cases that we see, are due primarily to what I call divergence excess. This condition is differentiated from a convergence insufficiency by the accompanying signs given in parallel columns.

### DIFFERENTIATION OF CONVERGENCE INSUFFICIENCY AND DIVERGENCE EXCESS

| CONVERGENCE INSUFFICIENCY | DIVERGENCE EXCESS |
|---|---|
| Exophoria for distance slight (only one or two centrads). | Exophoria for distance marked (from 5 to 30 or more centrads). |
| Exophoria for near marked (8 to 30 centrads). | Exophoria for near moderate (much less than for distance) may be almost nil. |
| Prism divergence not excessive (4 to 8 centrads). | Prism divergence excessive (10 to 30 centrads). |
| Prism convergence deficient and difficult to increase by exercise. | Prism convergence normal or readily brought to normal after two or three attempts. |
| Convergence near-point remote (over 9 cm. from intercentral base line). | Convergence near-point normal. |
| Crossed diplopia, if present, marked for near points and increasing as object is brought toward eyes. | Crossed diplopia if present, marked for distance. Usually none for near. |

In both, rotations of eyes inward and outward in performing parallel movements normal and comitant; crossed diplopia, if present, not increasing to right or left, but increasing somewhat in looking up and decreasing somewhat in looking down.

*Insufficiency of an Adductor.*—From insufficiency of an adductor (internal rectus, superior and inferior recti), convergence insufficiency is differentiated by the fact that in the former condition either the power of movement of the affected eye inward is obviously impaired, or at all events the exophoria and the crossed diplopia, if present, increase definitely as the eyes are carried to one side or the other.[6]

### MIXED AND SPECIAL FORMS

In many cases a convergence insufficiency is combined with other conditions, and sometimes the picture is so complicated as to render the diagnosis difficult. The most common combinations are:

1. *Convergence Insufficiency with Divergence Excess.*—This combination is very frequent. In fact, in the regular development of an ordinary divergent squint, what generally takes place is that either a primary convergence insufficiency has superadded to it a secondary divergence excess, or vice versa. In the former case, we see first an exophoria for near with normal relations for distance, later a squint for near combined with an exophoria for distances, and finally a squint for both distance and near with total absence of converging power. When the divergence excess is the primary condition, we have first an exophoria for distance with normal relations for near. This condition may last indefinitely or at least for many years without change. Then there develops an exophoria for near also, and a squint for distance, and ultimately a squint at all ranges, with, however, often at least partial retention of converging power. The last-mentioned is shown by the presence of a relative near-point of convergence. Whichever condition is the primary one, the final picture presented by the combined conditions is then a squint marked for both distance and near, and great, though by no means always total, loss of converging power.

2. *Convergence Insufficiency with Secondary Weakness of Interni.*—Secondary weakness of the interni often develops in cases of divergent squint of long standing; that is, as a result of a continued insufficiency of convergence combined with a divergence excess. In this case the power of rotation inward in making parallel movements of the eyes to right and left is obviously diminished.

3. *Convergence Insufficiency with Insufficiency of the Elevators or Depressors.*—This condition, which

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[6] Increase from the midpoint to both sides if both interni are weak.

is manifested by a vertical deviation, increasing characteristically in some special quarter of the field of fixation, is not infrequent. In some cases of the sort the convergence insufficiency is more apparent than real (see below in the consideration of etiology).

4. *Convergence Insufficiency with Divergence Insufficiency.*—This rare and troublesome condition is marked by an exophoria for distance with very low prism divergence or actual homonymous diplopia for distance, and by marked exophoria for near with remote near-point of convergence. In some extreme cases there is an actual paresis of both divergence and convergence (see below in the section on convergence paresis).

5. *Anomalous Cases.*—Cases of rather peculiar physiognomy are those in which (*a*) there is a marked exophoria for near (10 or 12 centrads) with normal relations for distance and with normal convergence near-point, and (*b*) those with very slight exophoria for near but with remote convergence near-point. The former are perhaps to be explained as due to a slight weakness of the interni *per se,* and the latter as an ordinary convergence insufficiency, in which the patient by special effort at times overcomes the deviation.

## ETIOLOGY

Convergence insufficiency is either primary or secondary.

*A. Primary Convergence Insufficiency.*—This may be either accommodative or non-accommodative.

1. *Accommodative* convergence insufficiency is the form in which the patient converges insufficiently because he accommodates insufficiently. This form is observed, first, in uncorrected myopes who, as they do not have to accommodate in looking at near-points, so do not converge either; second, in corrected presbyopes and hyperopes who, when they put on convex glasses, find themselves enabled to read without exerting their usual quantum of accommodation and, as they let their accommodation relax, relax their convergence also.

In all these cases of accommodative convergence insufficiency it is evident that the patient really has full power of accommodation, but, owing to refractive conditions of one kind or another, he exerts only part of it. As he puts only a portion of his available force into his accommodative work, he also puts only a portion into the work of converging and hence converges imperfectly. But there is another class of cases, rather frequent, in which the accommodation is really subnormal, and the patient works it to its limit, putting into it all the nerve-power at his command. How he will converge in this case will depend on the cause of the weak accommodation. There are two possibilities. Either the patient's neuromuscular accommodative apparatus is defective and his nerve-power is normal, in which case he makes a tremendous effort and nevertheless accomplishes very little, or his accommodative apparatus is normal but the nerve-power supplied to it is inhibited in some way, in which case he makes but little effort and therefore accomplishes but little. In the former case there is overstimulation, in the latter case understimulation of the accommodative apparatus and hence also of the converging apparatus. If the converging apparatus is normal, the patient should in the first case have a convergence excess, and in the second case a convergence insufficiency as the result of a genuine accommodative weakness. Examples of these two kinds of association do, in fact, occur, but are not very common, so that accommodative convergence insufficiency is apparently not often due to accommodative weakness of central origin. It more frequently happens that the two conditions, instead of being dependent one on the other, occur simultaneously, both being due independently to the same cause. In this case, of course, the convergence insufficiency is non-accommodative in origin.

Akin to accommodative convergence insufficiency is what we may call the *visual* form in which, owing to indistinctness of vision in one or both eyes or to incorrigible vertical diplopia, the patient has little impulse to converge. This is found in uncorrected presbyopia and in hyperopia of high degree; in anisometropia (very common); in poor vision due to opacities in media and also in absolute blindness of one eye, and in hyperphoria or other muscular anomalies which render fusion difficult. In cases of this sort the convergence insufficiency is usually spurious, at least at the outset. The patient, that is, can converge perfectly, but has no inducement to do so because the diplopia produced by the failure to converge is no more confusing than is the patient's vision when he converges well and superposes the two visual images. In other words—and this holds for most of the cases of the accommodative form too—the condition at the beginning is rather a convergence disuse than a convergency insufficiency. Later, however, continued disuse develops an actual insufficiency.

2. The *non-accommodative* form comprises about 60 per cent. of all cases of primary convergence insufficiency. It includes all those cases in which the refraction and accommodation cannot in any way be held accountable for the genesis of the motor anomaly. This is the case in patients who have low degrees of hypermetropia and astigmatism with equal refraction and with good vision in both eyes; also in patients who, though myopic, have been wearing their myopic correction for a long time, so that they are using their accommodation quite like emmetropes. In all these cases it is noted that continuous wearing of the proper refractive correction does not affect the muscular deviation except possibly to increase it. In a true accommodative convergence insufficiency, on the other hand, the deviation is diminished or even abolished altogether by the continuous use of the right glasses.

Non-accommodative convergence insufficiency is due to (*a*) conditions causing general nervous or systemic depression (neurasthenia, hysteria, anemia, convalescence from exhausting illness, and the like) ; (*b*) nasal obstruction (quite common), and (*c*) some unknown cause acting in persons whose muscular power and coordination otherwise seem normal. The more serious and inveterate cases, and particularly those that approximate to a convergence paresis in character, are perhaps due to a localized lesion, congenital or acquired, of the central nervous system (see below, section on convergence paresis).

As already noted, a convergence insufficiency, even when non-accommodative, may be associated with a weakness of accommodation which, like the insufficiency itself, is of central origin, the two being due independently to the same cause.

3. *Combined Accommodative and Non-Accommodative Forms.*—These mixed forms are not uncommon.

Thus a convergence insufficiency, itself non-accommodative, may be increased and its symptoms may be intensified by the introduction of an accommodative element. This happens, for example, when we give a hyperope or a presbyope who already has a convergence insufficiency, too strong a glass for reading.

*B. Secondary Convergence Insufficiency.*—When a divergence excess has lasted a long time, a secondary convergence insufficiency often develops, producing the combined form of deviation, the characters of which have already been noted. (See for further particulars remarks on "Course," below.)

### CONVERGENCE PARESIS

No absolute line of demarcation distinguishes a convergence insufficiency from a convergence paresis. In fact, the extreme form of convergence insufficiency that we see in a well-marked periodic divergent squint is really a convergence paresis more or less obscured by complicating conditions. The term "convergence paresis," however, is usually applied to a condition often sudden in development and evidently due to localized lesion or functional disturbance of the nerve centers, marked by the following features:

1. In far distance (points beyond 20 or sometimes beyond 50 feet), little or no deviation.

2. The moment an attempt at convergence is made, that is, as soon as the patient tries to follow an object brought from the 20-foot distance toward the eyes, exophoria and crossed diplopia set in and increase *pari passu* as the object is approximated. The increase takes place with almost mathematical regularity.

3. The diplopia once evident is almost or quite insuperable. Thus, when even it is so small that it requires only a 2- or 3-centrad prism to correct it, the patient is usually quite unable to correct it by voluntary effort. So, too, at a distance at which he normally sees single, he is quite unable to overcome even a 2- or 3-centrad prism, base out.

4. No attempt is made at convergence. When an object is carried toward the eyes, one turns toward it and the other turns off, *pari passu*. In a pure convergence insufficiency, however great, there is usually at least an attempt at convergence.

5. Just as in ordinary convergence insufficiency, so also in convergence paresis the lateral movements of the eyes are unaffected, each eye as it moves with its fellow to right or left rotating quite normally inward. So also the crossed diplopia, taken at any given distance, does not increase as the eyes are carried to the right or left. In other words, we are not dealing here with any paresis of the interni *per se.*

6. The convergence reaction of the pupil is usually absent.

Convergence paresis is rather rare. Still rarer is its combination with a paresis of divergence. In this condition, there is a single vision only at one definite point. Beyond this there is homonymous diplopia, within this crossed diplopia. Looking at an object situated at the point itself, the patient cannot overcome a prism, however weak, either base out or base in. It seems, in fact, as if his eyes were fixed immovably on a single point. But when it comes to making lateral or up and down movements, each eye moves as freely as usual and always in harmony with the other.

Convergence paresis may also be associated with paralysis of up-and-down movements of the eyes. It is at times, too, accompanied by paresis of accommodation, the lesions in this case being strictly coordinate; that is, they occur simultaneously as the result of the same cause and are in no sense dependent the one on the other. The picture then presented is very striking.

Convergence paresis is doubtless due to some lesion of the central nervous system. It occurs in multiple sclerosis (in 3 per cent. of the cases according to Uhthoff) and occasionally in tabes, and in isolated cases it is the result of traumatism, hysteria, tumor, hemorrhage and developmental disorders arising from affections of the ductless glands. It is also found as a congenital anomaly in most cases of the peculiar syndrome marked by paralysis of the external rectus, restriction of power of the internal rectus and retraction movements of the eyes.

In a case of mine which was associated with a marked paresis of accommodation, the voluntary dissociation of the eyes produced by prolonged work with the microscope seemed to be the starting-point of the trouble. In some cases we must admit that the etiology is quite obscure.

### COURSE

An accommodative convergence insufficiency of the ordinary type is, as I have noted, at the outset simply a convergence disuse. That is, the converging power is normally strong but is not used to its full extent. After a time, owing to this continued disuse, the converging power becomes actually subnormal. When this process is once initiated, there is a tendency, although by no means an invariable tendency, toward increase.

If it increase to a considerable point and is long maintained, it is then regularly followed by a divergence excess. Similarly, a divergence excess which has lasted a long time is likely to be followed by a convergence insufficiency. Thus result the combined forms (see above "Mixed and Special Forms"), which constitute the ordinary form of a fully developed divergent squint. A divergent squint, in other words, usually develops in one of the two following ways:

1. An accommodative or at all events a visual convergence insufficiency develops out of a convergence disuse. This at first is a heterophoria, which is marked for near points only. Gradually this increases by the superadding of a divergence excess, and now there is a heterophoria for a distance with a divergent squint for near (direct periodic squint). The deviation at both distances increases until ultimately there is a squint both for distance and near (continuous squint), but more marked for near.

2. A divergence excess, which produces a heterophoria marked for distance and but slight for near, which may last unchanged for years, ultimately increases by the superadding of a convergence insufficiency. There is now a divergent squint for distance and a heterophoria for near (inverse periodic squint). These increase until ultimately there is a squint for both distance and near (continuous squint), more marked, however, for distance than for near.

In both cases, a further change that takes place after a while is a weakening of the interni (hitherto normal) and a contracture or hypertrophy of the externi. The inward rotations are then no longer normal and the deviation becomes greater still.

The course of a non-accommodative convergence insufficiency—and the same is true of a convergence paresis—depends on its causation. If due to a temporary condition, the insufficiency or the paresis will clear up; otherwise it may remain unchanged for years or in some cases gradually increase. A non-accommodative convergence insufficiency may thus also give rise to a divergent squint.

### SYMPTOMS

The symptoms of convergence insufficiency are diplopia (with accompanying confusion and vertigo), asthenopia, eyeache, headache, and sometimes more remote reflex symptoms.

Diplopia, although it can almost always be elicited, is rarely a subject of spontaneous complaint except in convergence paresis, when it is often very distressing. Confusion (especially in reading) and vertigo may occur, but are not common except in extreme cases of insufficiency or cases of actual convergence paresis.

Asthenopia is a common symptom, and often is associated with burning and irritation of the eyes (asthenopia irritans) or with eyeache (asthenopia dolens). The asthenopia is sometimes extreme and incapacitating. It is often aggravated by the use of convex glasses, because with them the patient not only tends to relax his accommodation and hence increases his convergence insufficiency, but also brings objects close and so puts more strain on his convergence. Sometimes, in fact, the asthenopia is so great that the patient cannot wear the proper glasses at all.

Headache is not a very common symptom, nor are reflex symptoms very often encountered—not so often, indeed, as in other muscular anomalies. Of course when squint has developed, the deformity due to it constitutes for the patient a symptom of considerable moment, and in convergence paresis and in convergence insufficiency due to general conditions we have to reckon with the symptoms arising from the causal lesion. So, too, we have to reckon with the asthenopic symptoms due to the associated insufficiency of accommodation when this is present. These may be difficult to distinguish from the symptoms of convergence insufficiency itself.

In a large proportion of cases of convergence insufficiency there are no symptoms whatever that can be referred to the motor anomaly itself, that is, that are not relieved simply by treatment of the attendant conditions (refractive errors, etc.).

### TREATMENT

The measures available for treating a case of convergence insufficiency are:

1. Correction of the refraction.
2. Correction of exciting causes other than refractive errors.
3. Practice in converging on an approximating point and with prisms, base out.
4. Practice with the amblyoscope, stereoscope and bar-reading.
5. Practice in the recognition of diplopia with the aid of the red glass, etc.
6. Prisms for constant wear.
7. Operation.

1. In a case of convergence insufficiency the first demand of treatment is to correct the *refraction* with scrupulous care. By so doing we remove any accommodative or visual element which may aid to produce the insufficiency, and thus in a moderate proportion of cases relieve or at least diminish the tendency to deviation. In a much larger proportion of cases, the correction of the refraction, while not producing much effect on the deviation itself, does relieve the symptoms.

A convergence insufficiency associated with hyperopia is sometimes aggravated by the full correction of the refractive error. In such cases we may have to undercorrect the hyperopia by an amount which will vary with the conditions and symptoms present in the given case, but which in general will have to be determined experimentally.

In presbyopia associated with convergence insufficiency it is particularly important not to add too strong a glass for reading, since with such a glass the patient will have to bring objects too close and thus impose too great a strain on the already subnormal convergence. Neglect of this precaution is a frequent cause of trouble in giving glasses to presbyopes, especially in the early stages of presbyopia.

As in other muscular conditions, so also especially in convergence insufficiency, correction of the refraction should be the first means used, and until its efficacy has been tested by long and persistent trial, other treatment should not generally be employed. Otherwise we shall fail to differentiate between the symptoms due to the refractive error and those due to the muscular trouble. By eliminating one set of symptoms at a time, and the one, moreover, that is usually of most significance to the patient, we shall be surer of our diagnosis and more likely to give ultimate relief. It need scarcely be added that the earlier in a case the refractive treatment can be applied the better the result we may expect.

2. In non-accomodative convergence insufficiency and particularly in convergence paresis, the exciting cause (neurasthenia, hysteria, nasal disease, organic disease of the nervous system) must be sought out and treated. In cases the etiology of which is not evident, tonics, open-air exercise and the restriction of near work for a season are often indicated.

3. Exercising the convergence by simply making the patient fix sharply with both eyes a dot brought steadily up toward the eyes or by practice with prisms, base out, is of service both in accommodative and in non-accommodative convergence insufficiency. I have elsewhere[7] laid down precise rules for what I believe to be the best method of making the practice. In general I think it important that in simple convergence insufficiency, exercise with converging prisms should be employed with the test-object at the reading distance only, while if there is an associated divergence excess the exercise should be done with the object both at 20 feet and at near. These exercises seem to me to succeed particularly well when there is an associated weakness of accommodation; the latter is also thereby stimulated.

4. The use of prisms for exercising the convergence presupposes ability to recognize diplopia and to fuse double images. If the patient is deficient in either regard, we shall secure better results with the amblyoscope. The way to use this is, first, to get the patient habituated to seeing with the two eyes together, that

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[7] Duane, Alexander: Ophth. Rec., June, 1911.

is, to set the instrument in such a way that he clearly makes out two distinct images. The second step is to habituate the patient to seeing with both eyes when the images are brought close together, and finally to adjust the tubes so that the two images are superimposed and actually fused. The third step is to make the patient practice convergence after the images are thus fused, that is, to urge him to swing the tubes toward each other while still maintaining fusion. The successful carrying out of these different stages of the practice may take weeks or months.

For the proper carrying out of this practice, the amblyoscope should have a suitably graduated arc, so as to measure the position of the tubes and register the amount gained by exercise, and also a vertical adjustment to correct the hyperphoria that is not infrequently present.

Fusion may also be exercised usefully with the stereoscope and with bar-reading.

5. Another way, besides the amblyoscope, of getting the patient to recognize the fact that he sees double and to pave the way to the use of converging prisms, is to separate the images widely by prisms, or to make the images dissimilar by using a red glass before one eye and a green glass before the other. Systematic exercises at home of this sort may usefully precede exercises with prisms, base out.

6. The use of prisms, base in, to correct the deviation is in my belief a procedure to be condemned. Affording a temporary relief, it has seemed to me that in most cases such prisms ultimately increase the deviation and the symptoms, too. On the other hand, the correction of a coexistent hyperophoria by prisms, base up or down, may afford considerable relief to the symptoms and make the lateral deviation much more manageable by ordinary means.

7. Operation should not be done unless all other means have been thoroughly tried and have failed and unless also the symptoms are severe and likely to be permanent. In a simple convergence insufficiency, advancement or tucking of the internal recti is indicated. It may or may not be combined with tenotomy of the external rectus, but if we are dealing with a simple convergence insufficiency, the tenotomy should

not involve the lateral attachments of the tendon and should be done to such an extent only as to relax the strain on the advanced muscle. If there is well-marked divergence excess, free tenotomy of the externus should be combined with the advancement of the internus.

It is best to operate on one eye and from four to six weeks later operate on the other if necessary.

If there is a considerable vertical deviation this should in general be relieved by operation before any attempt is made to operate for the lateral displacement. Otherwise the lateral displacement is liable to recur, and on the other hand, if the vertical deviation is corrected first, it may happen that the lateral deviation may not need correction at all.

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# MEMORANDA

# TREFOIL KERATECTOMY *

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Keratoplasty has been successfully employed to remedy certain corneal defects, but only a moiety of procedures have been devised to meet the indications present in anterior staphyloma. When a protruding but circumscribed corneal ectasia has proved to be more or less obstructive to the vision, and the remaining tissues of the globe have not been seriously involved in the pathologic process, conservative surgery demands that a mobile globe shall be preserved, and, whenever it is possible, that a modicum of useful vision shall be secured.

To meet these indications I devised, in 1895, a simple form of corneal excision, which I have designated as *trefoil keratectomy*. It is adapted chiefly to those cases in which a limited portion of the cornea is staphylomatous and in which correction of the ectasia can be made by the removal of a three-leaved flap from the lower part of the pendulous cornea (Fig. 1). When, however, the whole cornea is involved and a four-leaved flap must be excised to correct the deformity, the procedure is designated as *stellate keratectomy*.

## PRELIMINARY TREATMENT

Full antiseptic precautions should be observed, as most cases of corneal staphyloma are the result of infectious processes, such as trachoma, suppurative conjunctivitis and other mucopurulent discharges. A course of sedative treatment should, therefore, be applied to the palpebral conjunctiva. As many of these cases are complicated by lacrimal blennorrhea or obstruction, it is often necessary to perform rapid dilatation of the tear-duct. If this is indicated, it should be done at least one week prior to the corneal excision. It is often wise to irrigate the nose with an antiseptic wash.

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### ANESTHESIA

This operation has been performed under cocain-epinephrin anesthesia, but full ether narcosis is to be preferred, since the restlessness of the patient might

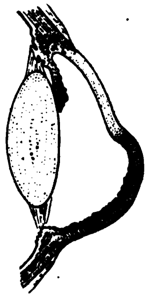

Fig. 1.—Vertical section of anterior staphyloma, showing clear cornea above and ectasia below. The thickened iris-tissue has been glued to the cornea by plastic inflammation.

interfere with the proper introduction of the sutures, or even cause an injury to a transparent lens, if such should be present.

### INSTRUMENTS REQUIRED

In my earlier operations I used a speculum, Graefe knife, fixation forceps, a pair of small, strong scissors, a needle-holder, needles and fine black silk sutures. In cases of emergency an iris forceps, iris scissors, silver spoon and spatula, and a wire loop may be required and should be included on the tray. In a later method I changed the technic by substituting a keratome for the Graefe knife, and a corneal punch (Fig. 2) instead of the scissors. The two methods appear to be equally efficient, although in certain cases the punch cannot be used because of lack of space in which to introduce it.

### DESCRIPTION OF OPERATION

Three methods will be described, (*a*) trefoil keratectomy with the scissors, (*b*) trefoil keratectomy with the punch and (*c*) stellate keratectomy with both scissors and punch.

### *Trefoil Keratectomy*

*Scissors Method.*—First Stage: With the speculum inserted and the globe firmly held by fixation forceps, the operator should first inspect and outline the exact area to be excised (Fig. 3 *A*). The Graefe knife is then passed horizontally through the base of the staphyloma from the temporal to the nasal side, keeping the edge of the blade turned directly forward, as for a Saemisch incision, and drawing it backward and forward but not allowing it to emerge fully until the aqueous fluid has completely escaped (Fig. 3 *B*).

Second Stage: One blade of the scissors is now introduced beneath the upper margin of the horizontal incision, a little to the right of the center (2 or 3 mm.), and a long vertical incision made. A second converging cut is then made, the same distance to the left of the center, which joins the first at the apex and thus makes a V-shaped incision and opening (Fig. 3 *C*).

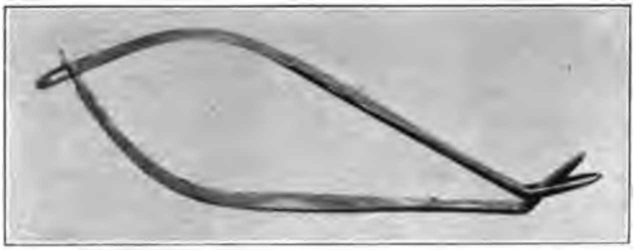

Fig. 2.—Corneal punch, with long oval points. The spring was adapted from McClure's iris scissors.

Third Stage: The two corners of the opposing wound-margins are successively grasped with the forceps and a triangular piece (Fig. 3 *C*, upper margins) excised by scissors from each corner, thus making a three-leaved opening (Fig. 3 *D*).

Fourth Stage: The vertical wound is closed by two or three fine black silk sutures passed through the opposing wound-margins and tied (Fig. 3 *E*). The horizontal wound is then sutured in like manner (Fig. 3 *F*). A Kalt suture (linen) may, if preferred, be inserted through the outer layers of the cornea and tied as a mattress-suture. The cut surfaces must be carefully approximated at all points.

*Punch Method.*—First Stage: A vertical incision is made through the base of the staphyloma with an angular keratome (Fig. 3 *G*), and the inferior blade of the punch is passed into the anterior chamber and beneath the cornea, while the punch is closed with a short, quick thrust, thus excising the left lateral flap (Fig. 3 *G*, shaded part).

Second Stage: The punch is removed, introduced on the other side of the wound-edge, and the right lateral flap is excised in the same way (Fig. 3 *H*, shaded part).

Third Stage: The upper margin of this long, oval wound is now grasped at its center by the punch and a vertical piece excised (Fig. 3 *I*, shaded part), leaving a trefoil opening (Fig. 3 *D*).

Fourth Stage: The sutures are now inserted to close first the vertical wound (Fig. 3 *E*) and then the horizontal wound (Fig. 3 *F*), as previously described.

### *Stellate Keratectomy*

This operation is chiefly indicated when the staphyloma involves the whole of the cornea, or when a central mushroom staphyloma has developed. It can be performed either with the knife and scissors or with the keratome and punch, as is preferred.

First Stage: A horizontal incision is made in the center of the cornea, with the Graefe knife or with the keratome (Fig. 3 *J*).

Second Stage: The upper and lower vertical flaps are excised with the scissors or with the punch (Fig. 3 *J*).

Third Stage: In like manner the two horizontal flaps are excised (Fig. 3 *K*).

Fourth Stage: Each section of the stellate[1] incision is closed by a single suture, and a central mattress-suture is finally inserted.

### COMPLICATIONS

If, by chance, abnormally high tension should cause extrusion of the lens, or if vision can be improved by the removal of a cataractous lens, the loop or the silver spoon should be used for this purpose and the toilet of the wound made before tying the sutures. If it seems possible to improve the vision by iridectomy or by De Wecker's irito-ectomy, this may be done

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[1] Since preparing this paper my attention has been called to the star-shaped incision of Attias, recently devised and published in the Ophthalmoscope, January, 1914. His sutures cross over from the upper right flap to the lower left flap and vice versa.

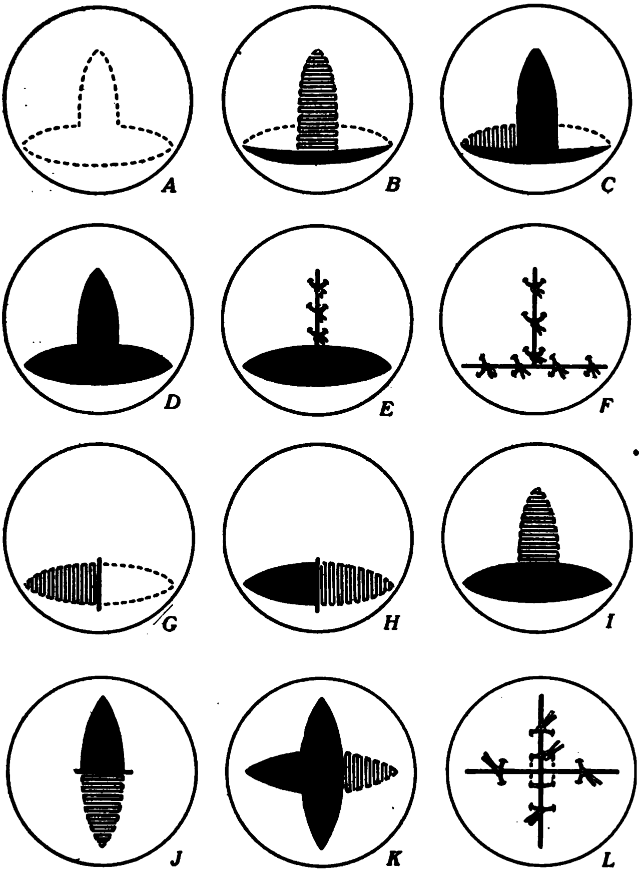

**Fig. 3.—Incisions and sutures for both trefoil and stellate keratectomy; *A,* three-leaved flaps outlined; *B,* corneal incision below, *à la* Saemisch, shaded part of upper flap ready for removal; *C,* upper flap excised, left tip shaded; *D,* trefoil keratectomy completed; *E,* vertical wound sutured; *F,* both vertical and horizontal wounds sutured; *G,* vertical keratome incision for punch operation, left flap shaded; *H,* left flap excised, right shaded; *I,* right and left flaps excised, lower one shaded; *J,* horizontal keratome incision for stellate keratectomy, upper flap excised, lower one shaded; *K,* upper, lower and left flaps excised, right flap shaded for removal; *L,* stellate keratectomy completed and sutures inserted.**

before the wound is closed, but *pari passu,* it is better to postpone such an attempt until after healing has occurred.

### POSTOPERATIVE TREATMENT

Mercuric chlorid ointment should be inserted into the conjunctival culdesac and a light bandage with a cresol solution compress adjusted. A slow-healing wound may be stimulated by touching it with a weak solution of silver nitrate. An infected wound should have an application of liquor formaldehydi, 0.5 per cent., and ice-pads should be applied. A wound that slightly gapes or shows other irregularity should receive a gentle touch with the hot galvanocautery point.

### REPORT OF CASES

I shall briefly cite three interesting examples of this keratoplastic procedure in types that are quite diverse, the first occurring in an extremely attenuated cornea that had yielded to a superficial necrosis through the erosive action of a blennorrhea, the second in a cornea that was practically normal except in the ectatic area involved, and the third in a cornea that was greatly thickened by plastic deposit and made staphylomatous by the increased intra-ocular tension behind it.

Case 1.—Margaret M., aged 19, was first seen by me during an attack of typhoid fever, March 17, 1895, in consultation with Dr. George Morley Marshall, the present otologist at St. Joseph's Hospital, who was at that time in charge of the medical service. There was primary infection of the ethmoid and antrum, with secondary involvement of the ears and eyes, the former through an infectious otitis media purulenta and the latter through a profuse blennorrhea of the lacrimal sac, which kept both eyes bathed in a purulent discharge in spite of free irrigation and ice-padding. The left eye showed multiple areas of corneal ulceration with some ectasia. The cornea of the right eye was markedly involved by sloughing keratitis. One week later it was found necessary to perform a Saemish incision in the right eye to check the corneal slough, but the globe proceeded to shrink and was finally enucleated, June 10, 1895, at which time rapid dilatation of both tear-ducts was performed, *ad maximum.*

Oct. 28, 1895, an examination of the left eye showed adherent leukoma with marked anterior staphyloma, the ectasia being very thin and bulging forward below, as illustrated in Figure 1, while a small area of clear cornea was still preserved along the upper margin. The eyelids could not be closed over the staphyloma. The vision was limited to light perception and light projection. There was no discharge present.

As the patient had been able to count fingers before the pupil was drawn into the scar, and as the globe seemed to be healthy, I decided to make a plastic operation on the cornea. Ether was administered and trefoil keratectomy was performed, the primary incision being made with a Graefe knife through the base of the staphyloma, after which a large vertical flap was cut out with the scissors and the two lateral tips later excised. The iris was freed from above. The corneal wound was closed by fine black silk sutures, the vertical first, and the horizontal last. Healing was uneventful and the cornea became nicely flattened. Vision had recovered to the degree of 10/200 at the time of patient's discharge from the hospital.

Case 2.—Mary McC., aged 19, applied to Wills Eye Hospital March 20, 1908, suffering from corneal staphyloma of the right eye, down and in. Scar was somewhat weakened at lower margin. Cornea was clear above. The globe was not involved. Tension + 1. Iris was adherent to the scar. The lesion was the result of a blow received during childhood. Iridectomy was performed by me four years ago.

April 24, 1908, patient was admitted to the hospital during the internship of Dr. J. Milton Griscom, at present assistant surgeon to the hospital, to whom I am indebted for these notes. Trefoil keratectomy was performed and the wound sutured.

May 1, 1908, sutures were removed.

May 3, 1908, shallow anterior chamber, due to a small break in the line of the wound.

May 5, 1908, deep anterior chamber. Wound closed.

May 20, 1908, patient was discharged cured. Cornea was firm and flattende. Vision 20/100. The present appearance of the right eye is well shown in Figure 4.

Case 3.—Alphonso R., miner, aged 47, came to Wills Eye Hospital, Dec. 4 ,1908, with ulcerative keratitis and adherent leukoma, the result of severe traumatism from a flying piece of steel, six weeks before. Perforating ulcer, 3 by 4 mm.

Dec. 6, 1908, Stilling's operation with lead style and rapid dilatation of tear-duct was performed to eliminate the danger of lacrimal infection.

Jan. 13, 1909, iridectomy was made for visual purposes.

Jan. 29, 1909, patient was discharged without visual improvement.

July 12, 1909, patient returned with large staphyloma of cornea at the site of the old scar. Marked interstitial thickening of the cornea, which resembled leather in looks and

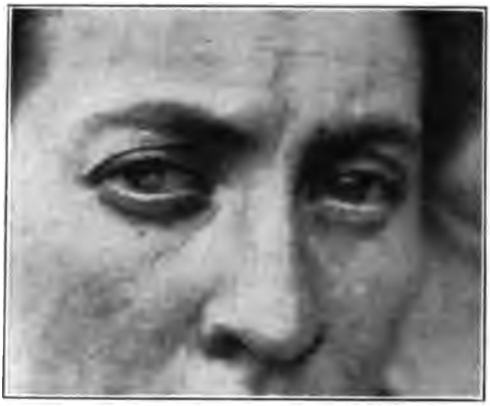

Fig. 4 (Case 2).—Result of keratectomy in the right eye; clear cornea above; flat leukoma below; final vision 20/200.

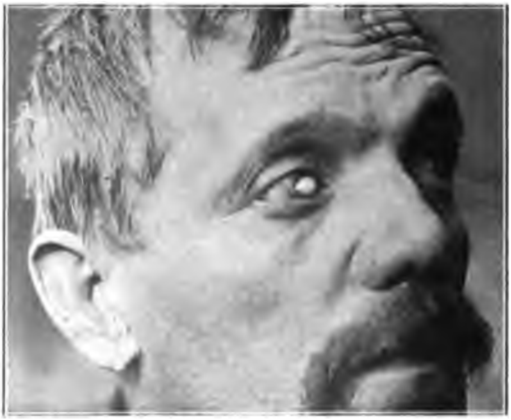

Fig. 5 (Case 3).—Large leathery staphyloma excised in the right eye, leaving a well-flattened cornea.

feeling. Tension was + 2. Under ether anesthesia three triangular areas were excised with the corneal punch (trefoil keratectomy) and the necessary sutures inserted. The sudden release of intra-ocular pressure caused the lens to be extruded, but no other complication ensued.

Aug. 20, 1909: Recovery was uneventful. Patient was discharged with a well-flattened scar but no improvement in vision. Figure 5 shows the appearance of the eye at that time.

1625 Walnut Street.

# MEMORANDA

# FURTHER OBSERVATIONS ON THE HEMIOPIC PUPILLARY REACTION OBTAINED WITH A NEW CLINICAL INSTRUMENT *

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The need of such a test as the hemiopic pupillary reaction, if it holds in cerebral cases any of the topical diagnostic possibilities originally suggested by Heddaeus and later elaborated by Wernicke,[1] is so great that no labor should be spared fully to estimate the value of every promise it may offer.

In a paper[2] before this Section last year a complete historical summary and bibliography were given of this subject and of the Wilbrand test.[3]

### PREVIOUS OBSERVATIONS

It will suffice here to recall some cardinal points from the researches of previous observers. Most important was the work of Hess in clearly showing what a great stumbling-block the phenomenon of dispersion light within the eye media had been in both making and interpreting observations, and, since it has been frequently noticed in discussion that this

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* From the surgical clinic of Prof. Harvey Cushing at the Peter Bent Brigham Hospital, Harvard Medical School.

[1] In the Wernicke reaction, as the phenomena was later commonly designated, a slender beam of light was thrown on the blind and then on the seeing retina of a hemianopic eye. Briefly, if there was no difference in pupillary reaction the lesion was central or posterior to the corpora quadrageminata and the reflex arc. While if there was a difference in reaction, the lesion was anterior or peripheral, interrupting the reflex arc from the blind retina to the sphincta iris.

[2] Walker, C. B.: Topical Diagnostic Value of the Hemiopic Pupillary Reaction and the Wilbrand Hemianopic Prism Phenomenon, with a New Method of Performing the Latter, THE JOURNAL A. M. A., Sept. 27, 1913, p. 1152.

[3] The results of further investigation of the latter subject are too space-consuming to be included in this report, but will be given later.

phenomenon is still apparently a hazy concept to many, it may be worth while to dwell on it for a moment. By dispersion light is understood that light which in traveling the media of the eye is diverted by diffraction, refraction, etc., in all directions from the rectilinear path of the ray entering the eye. Dispersion light is strongest immediately about the image on the retina and decreases farther away from the image, but is still strong enough at right angles to the axis of the ray to render the path of the ray visible, if observed in a dark room through a trephined scleral window in the freshly enucleated eye. A somewhat similar phenomenon is commonly observed when a searchlight is seen playing about in different directions on a dark night or when a beam of light passes through water, especially if any turbidity is present. Dispersion light may also be observed in one's own eye entopically when a beam of light is so arranged as to fall entirely on one's blind spot.

Hess, in one of his crucial experiments, placed a small light on a perimeter-like apparatus so that the light could be moved off or on the blind spot. The consensual pupillary reaction was noted by means of an entopic pupillometer on the other eye. He noted no pupillary reaction when the light was moved concentrically with the fixation point above or below the blind spot onto the seeing retina; indeed, sometimes there was a slight reaction in moving from retina onto blind spot due to reflection from the bright optic disk. If, however, the light was moved slightly toward the fixation point, a marked reaction resulted. Therefore he concluded that a small area about the macula contained all the pupillomotor fibers, so that when a ray of light was thrown on the peripheral retina, the resulting pupillary reaction was due to dispersion light stimulating the macula rather than to direct stimulation of the peripheral retina. Further, he showed that the exact center of the macula was the most sensitive point in the macular region. From a study of these facts it was at once obvious that previous observations were not at all well controlled with respect to dispersion light, the properties of which really constituted the crux of the whole situation.

This fact having been established, the next step was the production of a satisfactory instrument which would perform the test and at the same time control the dispersion light. The latter could not be dispensed with but could be made to affect the macula with constant strength in one of two ways: first, by rotating a light at fixed radius from the fixation point from blind to seeing field, and, secondly, in a "hemikinetic" manner, to use the term of Hess. This involves two equal areas of light, symmetrically disposed about the fixation point, and so arranged that each area may be exposed alternately as the other is closed. The intensity of the areas of life must be controllable about the point of photometric balance, and during the period of change the total area exposed must be constant and equal in amount to the full area of one of the lights. In this way the direction of the light entering the eye may be changed, but the amount of dispersion light reaching the macula remains constant.

Hess devised an instrument of sliding shutter type to accomplish this. Two equal vertical openings of rectangular shape equidistant from the fixation point were illuminated from behind through milk glass. The openings in the sliding shutter which exposed the two areas alternately were so placed that the moment one area began to be cut off the other area was exposed by the same amount. This fulfilled all the conditions except that of symmetry during the period of change, when light is cut off from one side of one area but added to the opposite side of the other area, as is of necessity the case with all plain sliding shutters. The result of this arrangement may be a pupillary jump during the change, since at that instant more dispersion light reaches the macula from one light by addition than is subtracted from the other light. This is most obvious in the mid position when the two half areas are found to be asymmetrically disposed with respect to the fixation point by exactly one-half the width of either area. Sliding shutters which will be light-tight have a tendency to stick at times and the resulting movement of the whole instrument is then such as to produce an oscillating series of asymmetrical positions. Further to minimize these objections, the areas have to be made as narrow as possible so that a pupillometer must be used to make observations satisfactorily, on account of the low intensity of direct illumination.

In order to obviate the difficulties of symmetry, the rotary shutter described in my last communication was devised and two or three hemiopic reactions were observed with it, but while it solved the problem of symmetry well enough, it was small, crudely made and poorly lighted as compared with the present instrument, so that a pupillometer was necessary to make observations in most cases.

### DIFFICULTIES WITH RECENT METHODS

This last point brings up a consideration of some of the difficulties encountered in making observations with the various forms of pupillometers. Questions of pupillary illumination or of observation ability on the part of the patient form a basis for most of these difficulties.

Thus in the entopic pupillometer the patient observes his own consensual pupillary reaction. A dispersion circle limited in size by the pupillary margins is projected on the retina by an encapsulated point source of light fixed close to the eye and with all extraneous light excluded. Any change in the size of the pupil is noticed as an enlargement or contraction of the retinal dispersion circle which is projected into space as a sort of moon or round window of light surrounded by darkness. While the patient notices this phenomenon and reports on it he must also keep his other eye carefully on the point of fixation and pay no attention to the shifting lights in the field of vision. If a field defect is present in both eyes the entire dispersion circle will not be seen except by special arrangement, such, for instance, as was used on the instrument presented last year.

While this method is the most sensitive we have, it has the common error of all monocular pupillometers of damping, as it were, pupillary dilatations, since as dilatation comes on more light is admitted to the eye, producing a tendency to contraction. Conversely, when pupillary contraction comes on, it is exaggerated, more light being excluded. But still more confusing is the peculiar pupillary bounce present at all times with most patients, and probably augmented by the preceding factors together with psychic or perhaps accommodative reflexes, although no change in light intensity has taken place. Altogether, the mental and physical condition of patients with field defects resulting from brain tumors is usually such as to inhibit the individual attention necessary for trustworthy observations with entopic method.

With the objective method, the monocular pupillometer being used, the main difficulty arises from the amount of light necessary to give a good view of the pupil. This degree of illumination is about the same in amount whether it be from the light in a pupillometer such as described in the previous paper,[2] or whether it be daylight or light from any source whatever, and the amount is such that as soon as the pupil is properly illuminated, it is at the same time markedly contracted. Under these conditions when a certain increment of light is added to the light already strongly contracting a pupil, the reaction may be hardly perceptible, while if the same increment of light is thrown on a dark adapted retina the reaction will be very noticeable. If the test lights used on the other eye are not of considerable strength, the stimulation may not be, in certain cases, sufficient to travel the consensual route and override the strong stimulation already present in the consensual eye sufficiently to give reactions that can be interpreted as other than equivocal or negative.

The logical sequence of these considerations was the desire to construct an instrument which would utilize the same light, necessary in any case for observation of the pupil, also to perform the hemiopic pupillary test. In addition to these requirements other desirable conditions to be fulfilled would be the possibility of varying the size of the areas symmetrically within all necessary limits and of rotating either area about the point of fixation from the blind to the seeing field. Further, both lights should be under complete control so that the intensity of either one could be varied above or below or made exactly equal to that of the other, or that the intensity of both could be varied together without disturbing the ratio of intensity between them. Such an instrument would also come much nearer satisfying the demands of a routine clinical instrument since the usual pupillometer trappings could be dispensed with, or used at will, and the pupillary reflex directly observed. Accordingly, somewhat over a year ago an instrument, of which a description follows, was constructed to meet, as well as possible, the imposed conditions.

### THE NEW INSTRUMENT

A diagram of the necessary light control is shown in Figure 1. The lights, *AA'*, are new 60-watt tungsten filament lamps selected from a large stock as nearly equal in all measurements as possible. From the wiring scheme it will be seen that they are connected in parallel. One side of each is directly connected to one side of the house current (110 volts). The other side of each lamp is likewise connected to the other side of the house current but must first travel through a set of resistances. The whole current first passes through a rheostat, *B*, whereby it may be varied in five steps from nothing to full strength. From the rheostat, the current then passes to the fixed bar of the sliding contact, *E*. This sliding contact, together with similar contacts *D* and *F*, may be moved laterally in contact with the edge of a slate slab *C* measuring 1 by 12 by 8 inches and wrapped with No. 26 B & S gage (0.016 inch diameter) German silver wire, the interval between the turns of

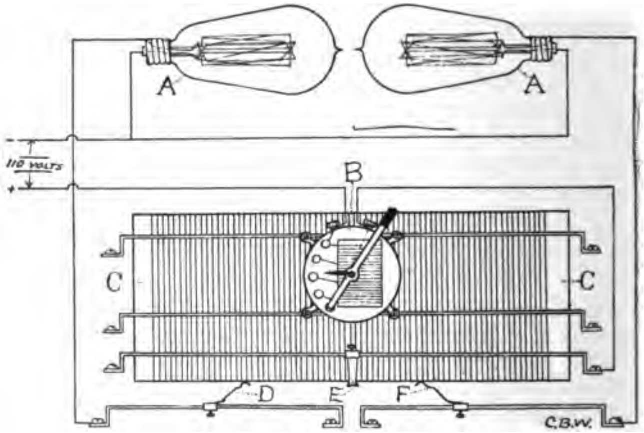

Fig. 1.—Diagram showing method of controlling lighting current in hemiopic pupillary apparatus: *AA'*, 60-watt tungsten lamps; *B*, rheostat controlling house current; *C*, slate slab wound with German silver wire; *D, E, F*, sliding contacts controlling current supplied to each lamp.

the coil being about 0.2 cm. This size of wire was found by experiment to have the necessary resistance to regulate the individual lamps, and still would not burn out under the contacts, with ordinary care. From the contact the current divides, passing through a variable amount of resistance to contacts *D* and *F*, and thence through their individual fixed bars, and on to their respective lamps. If now we wish to decrease the intensity of the light *A*, we have simply to move the contact *D* farther out or to decrease *A'* move the contact *F* farther out. If now with either of these contacts at any distance we wish to produce further change in the relative light intensity, we may simply move the contact *E* between the two contacts *D* and *F* until the desired point is reached without adding extra resistance but merely readjusting that already in use. Thus, if necessary, we can throw the whole or any part of the resistance on the slab *C* into the circuit of either light or divide it between them in any ratio. For purposes of measurement, the slab resistance is divided into ten divisions (points) each way from the center.

These two lights are encased end to end in the diameter of a heavy tin drum of about the size of an ordinary snare drum (14 inches diameter by 6 inches depth). Access to the interior is allowed by two doors in back. The inner surface of the drum is enameled white, and has two movable partition wings of the same curvature as the circumference so that when not in use they can be made to lie flat against the curvature of the drum. When it is desired to prevent one light from contributing more or less to the other, these wings may be turned inward to a corresponding degree or may be moved until they touch the axle for maximum effect. This axle in the center of the drum is hollow and acts as a hub posteriorly to receive the fixed axle on which the whole drum may be revolved, but anteriorly it has two large holes bored in it to transmit light to the fixation point, and still farther forward acts as an axle for the movable segments of the rotary shutter, which makes up the front of the drum.

The rotary shutter is an elaboration of the same type as that previously described. The segments

(Fig. 2) necessary to make up the shutter were cut out of heavy tin, although a similar weight in sheet brass would probably have worked up with less tendency to warp. The first disk, *F*, has two equal openings *L* and *L′*, the shape being such as to give the maximum area for the minimum movement of the shutter. The inner margin of each area is on an arc of 1 inch radius from the center and the outer margin on an arc of 5½ inches from the center, at maximum opening an arc of 45 being subtended. On the quadrants, *E* and *E′* is placed another segment of size and shape to fit the interval left by the next superimposed segment *G*. Thus a smooth surface is provided for the shutter to move on below, and above the proper level is obtained on which to solder an overhang under

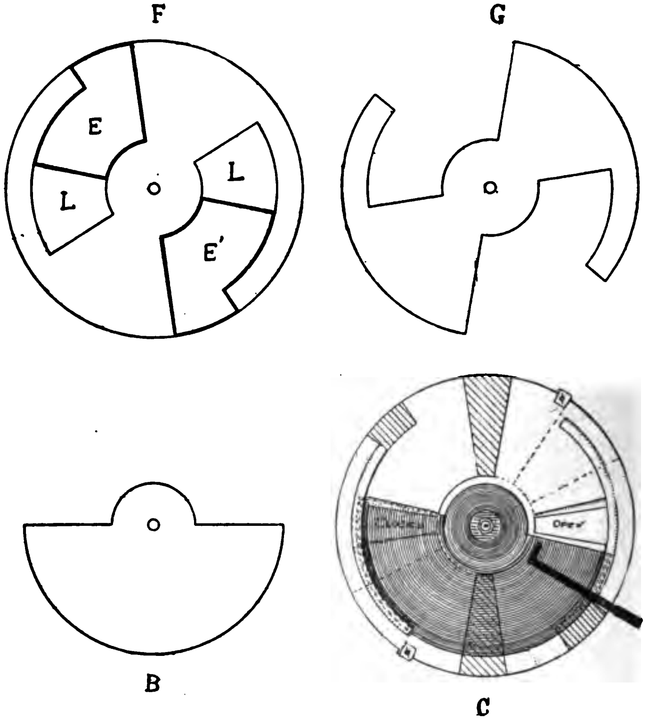

Fig. 2.—Plan of rotary shutter, assembled in *C* from segments *F*, *G* and *B*, in the position where the equal areas *L* and *L′* are cut down to about one-half full size.

which the shutter may pass and give a light-tight closing. The back of this disk carries two plates of milk glass covering the openings. The anterior end of the hollow axle also has a milk-glass window on which is marked a letter *A* and a dot, serving as a fixation point.

The next superimposed segment, *G*, serves mainly the purpose of symmetrically regulating the size of the lighted areas, but at the same time it carries two circular arms extending along the outer margins of the lighted areas, forming a smooth course on which the edge of the shutter may travel under cover of another overhang providing a light-tight margin. This disk is not fixed to the drum as the first one is, but is free to rotate when the clamps holding it to the first disk are loosened. The shutter is a semi-circular disk, *B*, actuated by a jointed hand lever, as is shown in the assembled shutter *C* drawn in the position where the lighted areas *L* and *L′* have been cut down to about one half their full size.

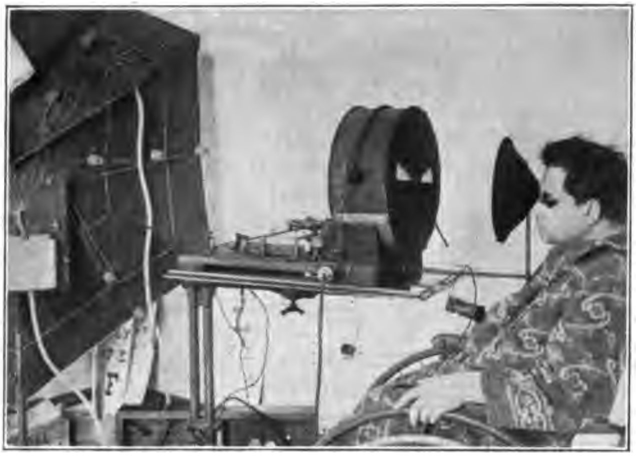

**Fig. 3.**—Patient in position before the hemiopic pupillary apparatus. At the left a portion of the new apparatus for performing the Wilbrand test on these patients may be seen.

To eliminate any asymmetry that may be due to reflection from the patient's face, a wide, short cone, truncated to fit the patient's eye, is provided on an adjustable support, so that the distance from the fixation point can be varied within any desirable limits, from 10 to 20 inches usually giving satisfactory illumination. A patient in position before the instrument is shown in Figure 3.

### METHOD OF EXAMINATION

Before making any tests, in order to avoid as much as possible any psychic or accommodative changes or any tendency to loss of fixation, the patient is familiarized with the movements of the shutter, until the shifting of light areas ceases to be of interest. The room is then darkened to exclude all asymmetrical light, and with the shutter at mid position or entirely open on the blind side, so that light adaptation may be kept equal, or as low as possible before the test is made, the patient is placed in position before the instrument with one eye covered, and the light is gradually increased until the movements of the iris can be accurately followed. This light strength varies slightly, depending on the color of the iris; it is greater for the darker irises. The patient is now told to sit back and close the eyes before proceeding with the observations, in order to increase the dark adaptation, which has a tendency to increase the difference in reaction, and to avoid fixation fatigue. Previously, of course, the lights have been photometrically balanced by means of the sliding contacts and further adjusted to the normal eye so that the pupillary reaction is the same from both lights.

Observation of direct pupillary reactions may be made with the unaided eye, or a short-focus telescope may be used. A very simple aid is made by rolling up a tube of stiff paper which will hold a magnifying glass in one and fit snugly about the observer's eye at the other end, thus at once cutting out shifting and somewhat confusing illumination on the observer's eye, and enlarging the pupillary movements. This tube may be held in the mid position to avoid asymmetrical reflections. Observation of the consensual pupillary reaction may be effected directly if all light from the instrument is screened off and limited to the eye under examination, by a heavy black conical curtain. Light is then admitted to the room until the movements of the consensual pupil can be followed, but without the use of the curtain the consensual pupillary reaction can also be observed objectively with the monocular pupillometer or subjectively with the entopic pupillometer previously described.

The rate of movement of the shutter has considerable effect on the resulting pupillary reaction. This holds true for the normal eye as well as for the hemianoptic eye. In general, on the normal eye if this change is made slowly from one light to the other no reaction may be noted if the accommodative and psychic reactions are not active. These latter reactions are very noticeable the first time a young person sits down to the instrument and simply fixes attention on the central point. If now the pupil is carefully observed with no change in light distribution one often notices in the course of the first minute or so decided variations in the size of the pupil. A noise or a spoken word may cause a distraction of attention or relaxation of accommodation with corresponding exaggeration of the normal hippus that may be noted for a time in absolute silence. These changes become reduced to a minimum as the patient becomes more and more at ease. In older persons whose accommodative reflexes are no longer active these reactions are much less noticeable if present at all. If a distant fixation point were observed through the hollow axle the condition would doubtless be better if it were not for the liability of the test lights near at hand to stimulate accommodative reactions as they flash into view.

When these reactions are quiescent there may be no pupillary reaction if the lights are slowly changed, but if the change is suddenly made, we notice a quick pupillary contraction followed by a somewhat slower dilatation to the former size. This may be repeated on the return change if the interval is two or three seconds or more. As the interval is decreased to the neighborhood of one second or less the pupillary contraction and dilatation becomes less until the interval is of such length that the pupillary reaction apparently does not have time to take place, either on account of the time required for transit of the reflex arc or for light adaptation phenomena. While it might be due to a combination of the two the interval is such as to favor light adaptation as an explanation. Symmetry of distribution on both sides of the macula is not necessary. The same thing happens if the lights are turned into the vertical position and a point a short distance out on the horizontal line through the center is taken as an eccentric fixation point. These reactions obtained on the normal eye are the same from either light when the instrument has been adjusted.[4] This same reaction may be elicited when the two lights are placed in the seeing field of a hemianoptic eye. But in any case, if the eye is allowed to fix a very short distance — a centimeter or less toward either light, for instance — so that asymmetry enters, a difference in pupillary reaction is of course noticeable.

In examining hemianopic cases the most satisfactory interval of alternation seems to be that which gives the maximum noticeable pupillary movement and seems to be about the same interval (2 or 3 seconds) as that at which the pupillary bounce is most active in the normal eye. The rate of change from one light to another is made as rapidly as possible for the same reason. In some cases in which the hemiopic pupillary reaction is marked, however, it is difficult to mask it with any kind of a shutter movement. Again, when the reaction is not marked, blinding the macula may be resorted to as a means of intercepting the reaction. A piece of heavy black paper having a circular hole an inch or two in diameter is held over one of the light areas and the patient told to look steadily at the center of this spot of light until looking at the center of the instrument fixation can still be maintained in spite of the blinding.

In order to get an idea of the magnitude of a hemiopic pupillary reaction, aside from measuring the pupillary sizes, two methods were used which gave relative results: first, the amount of resistance necessary to cut one light down to the point at which the reaction was the same from one dim light and one bright light. With the second method, the size of the areas was cut down until the consensual reaction could not be made out with the pupillometer on the other eye with any degree of certainty.

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[4] In all cases with eccentric fixation, the shutter movement must be as rapid as possible, for, although the total areas in the mid position of the shutter are symmetrical, there are two periods of asymmetry during the change before and after the mid position, but these give stimuli in opposite directions so that the total effect balances well with rapid motion.

In addition to the use of the shutter with the lights in fixed position one light may be exposed and the whole drum rotated so that the light may pass back and forth from blind to seeing field at constant distance from the point of fixation performing concentric rotation tests, as was mentioned in the last paper. Concentric rotation of one light entirely in the seeing field, however, may give a psychic as well as light adaptation contraction of the pupil; so that this movement must be repeated several times with patient in order to reduce the psychic element as much as possible by thorough familiarization with the procedure.

Cases with relative central scotomata and vision in the neighborhood of 20/200 can be assisted in fixation by sticking a white paper ring symmetrically around the fixation point. With this aid almost all cases can make out the center. At all events, if there is any tendency to look to one side of the center, it is of such direction as to reduce and not to increase a hemiopic pupillary reaction.

In the following cases, when pupillary measurements are mentioned in ordinary light, just sufficient room light is used so that the details of the pupillary margin may be seen when the patient is looking at a wall about 20 feet distant. The complete fields in these cases were taken with different-sized disks and other instruments in the manner described in a previous paper[5] but form and color fields for one-sized disk only will suffice here, since the full fields will be given elsewhere as a separate study. For the same reason and because of space requirements, the case histories are curtailed as much as possible.

In presenting the following cases those with anterior lesions will be given first. Of these, some have lost all vision in one eye and will be considered together as the second division of the first group.

CASE 1 (Surgical No. 663).—Interpeduncular teratoma. Dec. 8, 1913. Miss K. L., aged 11. Suffering with headaches for one and one-half years. Marked symptoms of dyspituitarism and internal hydrocephalus. Although left hemianopsia and left paresis were developing six months ago, fields could not be taken owing to dull mental condition, and it was not until after a decompression and a silver tube drainage for internal hydrocephalus had been performed that the mental condition cleared so that the fields (Fig. 4) were obtained at this, the fourth hospital admission.

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[5] Walker, C. B.: Some New Instruments for Measuring Visual-Field Defects, Arch. f. Ophth., 1912, xlii, No. 6.

*Eye Examination.*—No ptosis, exophthalmos or nystagmus; movements free and complete. Fundus of each eye: typical picture of low grade receded choked disk with very few evidences of secondary changes. Pupils react well to accommodation, direct and consensual light. Left pupil (6 mm. diameter) larger than right (5 mm. diameter). Fields and vision Figure 4.

*Hemiopic Pupillary Reactions.*—The direct pupillary test with central fixation on the rotary shutter showed a very marked and characteristic hemiopic pupillary reaction in both eyes. When the light was suddenly changed from the blind to the seeing field, there was a marked sudden contraction, followed at once by a slight dilatation. Equilibrium was quickly reached and when the light was changed to the blind field the pupil dilated considerably, and somewhat slowly, without any preliminary contraction or bounce whatever. On changing back to the seeing field the same contractive phenomenon was repeated, and further change to the blind field exhibited again the same dilatation and so on; the cycle could be repeated *ad libitum.* If the reaction lost intensity after several exposures, five or ten minutes in complete darkness again strengthened it. The reaction could be relatively measured for comparison with other cases by weakening the light in the seeing field until the reaction was the same from both lights. This required the sliding contact to be moved down to the point 9 before sufficient resistance had been introduced. Slow motion of the shutter gives about the same relative difference in pupillary size, but the little contractive

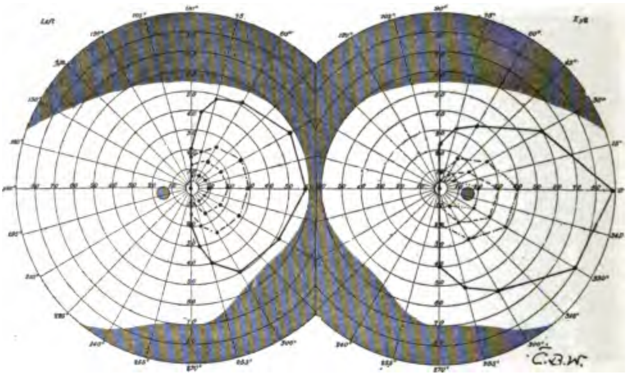

Fig. 4.—Case 1, tract hemianopsia. Hemiopic pupillary reaction marked. Vision in left eye 20/30; in right eye, 20/40.

bounce or rebound was absent. With the pupillometer on the other eye the reaction could still be made out when the areas were decreased to one-tenth or a little less, of the full area, and at best was not so marked as with direct examination. Slow rotation of one light about the fixation point from blind to seeing field gave about the same reaction as the slow shutter motion, but rapid rotation did not give so good a reaction as rapid shutter motion. With the lights placed vertically and eccentric fixation on the horizontal line through the fixation point so that both lights fell in the seeing field, rapid motion of the shutter gives a normal reaction from each light. With the lights in a horizontal axis and eccentric fixation in the vertical axis, so that one light is in the seeing, the other in the blind field the hemiopic pupillary reaction could be noted almost 40 degrees from the fixation point, but weakens rapidly as fixation is removed from the center. In all cases of eccentric fixation the shutter must be moved as rapidly as possible to reduce asymmetry during change to a minimum.

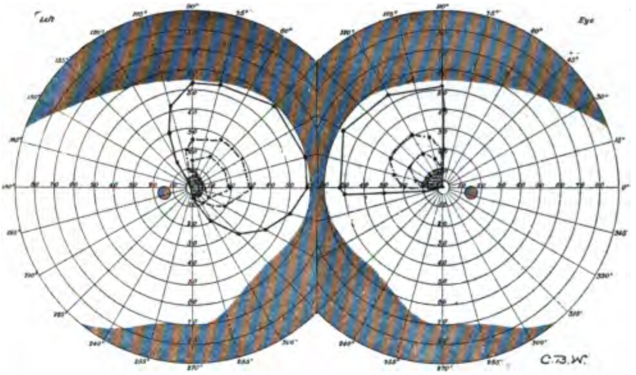

Fig. 5.—Case 2, chiasmal lesion. Hemiopic pupillary reaction marked. Vision in left eye, 20/100; in right eye, 16/200.

The tumor in this case was exposed at a subsequent operation lying in such a position as to involve the right optic tract.

Case 2 (Surgical No. 247).—Pituitary tumor. July 15, 1913. Mr. M. B., aged 43. Vision began to fail three years ago. Diplopia at same time lasting four months. Six months ago dizziness lasting two months. For past four years marked drowsiness during the day. Examination on admission shows a rather typical case of hypopituitarism.

*Eye Examination.*—No exophthalmos, nystagmus or muscular imbalance notable. Fundi show very pale optic disks with slightly blurred margins. Optic cup filled in and lamina cribrosa obscured apparently by a layer of new tissue which extends somewhat on to vascular trunks. Macular region and peripheral fundus clear. Pupils moderately dilated, 6 mm. diameter right, 5.5 mm. diameter left. Good reaction directly and consensually. Reaction to accommodation sluggish. Fields and vision Figure 5.

*Hemiopic Pupillary Reactions.*—Both eyes showed a well-marked hemiopic pupillary reaction on direct examination with the rotary shutter. The difficulty of central fixation was overcome by enlarging the fixation point with a white ring. The reaction measured 7 in the right eye and 8 in the left on resistance and could be followed consensually with the pupillometer to an opening one-sixth of the full area of lights. With symmetrical eccentric fixation and rapid shutter motion the reaction could be found along the line of field clearage to about 35 degrees from the macula before becoming equivocal. Rotation of one light concentric with the fixation point simulated the reaction obtained with the rotary shutter but could not be made quite as striking.

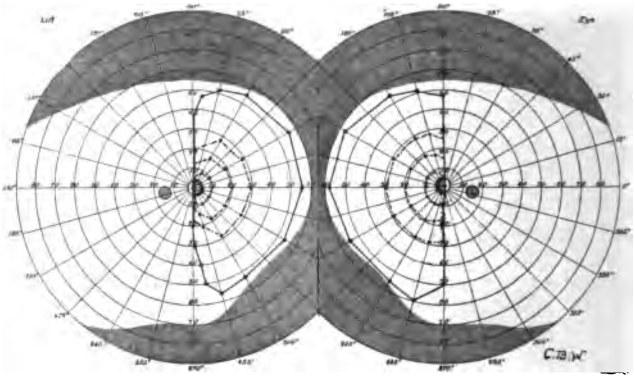

Fig. 6.—Case 3, chiasmal lesion. Hemiopic pupillary reaction well marked. Vision in left eye, 20/70—; in right eye, 20/70+.

Case 3 (Surgical No. 435).—Hypophyseal struma. Sept. 27, 1913. Mr. H. C. N., aged 42. Failing vision for two years associated with frequent headaches, diplopia, nausea and vomiting. Complete bitemporal hemianopsia for over one year. Examination on admission, typical hypophyseal struma with signs of neighborhood disturbances.

*Eye Examination.*—No nystagmus or exophthalmos. Transitory weakness of third nerve with pupillary inaction to direct light on right. Left pupil reacts well to direct light and consensually. Right pupil 3 mm. diameter, left, 4.5 mm. diameter in ordinary light. Fields and vision Figure 6.

*Hemiopic Pupillary Reaction.*—Well marked, measuring 7 on the resistance by direct examination of the left eye with central fixation on the rotary shutter. By fixation, eccentric in the vertical line, and rapid shutter motion, the reaction could be found as far as 25 degrees from the macula along the line of field cleavage. With eccentric horizontal fixation and lights in vertical meridian so that they fell on the seeing retina the reaction was the same as in the normal eye similarly tested. With the pupillometer the consensual reaction from the right eye was found also to be hemianoptic but not nearly so marked as in the left eye directly. Slow rotation of one light about the fixation point gave the same reaction as obtained with slow shutter motion, but rapid rotation of the light did not give as good a reaction as the rapid shutter motion.

Case 4 (Surgical No. 972).—Hypophyseal struma. Feb. 25, 1914. Mr. C. S., aged 49. Failing vision for eight months. No headache, nausea, vomiting or diplopia. On admission found to be well advanced case of hypopituitarism.

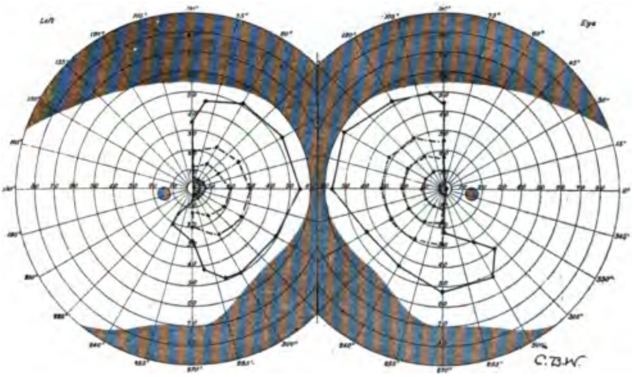

Fig. 7.—Case 4, chiasmal lesion. Well-marked hemiopic pupillary reaction. Vision in left eye, 20/100—; in right eye, 20/50+.

*Eye Examination.*—Slight tendency to exophthalmos; no nystagmus or diplopia. Movements free and complete. Fundus of Each Eye: Temporal portion of disk paler than normal; nasal portion shows little change. A few coarse stipplings of lamina cribrosa are visible in a wide shallow optic cup. Pupil in the left eye (5 mm.) slightly larger than the pupil in the right eye (4.5 mm.) in ordinary light. Pupils react well to direct and consensual light. Reaction to accommodation sluggish. Fields and vision Figure 7.

*Hemiopic Pupillary Reaction.*—Well developed in both eyes, slightly better in the left eye, measuring 6.5 to 7 on the resistance with rapid shutter movement. Rotary concentric test about the fixation point gave confirmatory reactions, but not as marked as with the shutter. With eccentric symmetrical fixation the reaction could be traced above along the line of field cleavage to 25 degrees. Below it soon became equivocal. With the pupillometer the consensual hemiopic pupillary reaction could be made out until the areas were cut down to one-quarter their full size.

Case 5 (Surgical No. 496).—Acromegaly. Oct. 16, 1913. Mr. C. M. W., aged 24. Very gradual failing of vision for three years. Diplopia first noticed two years ago and has become more persistent from that time. Signs of acromegaly first noted seven years ago. On admission patient found to be a typical case of acromegaly with incomplete bitemporal hemianopsia.

*Eye Examination.*—Patient is myopic 1 D. and uses monocle in the left eye to aid suppression of vision in the right eye, and avoid discomfort of diplopia, due to weakness of internal recti (2 degrees). No exophthalmos or nystagmus. Fundi show pale optic disks, right slightly paler than left, but lamina cribrosa is obscured in a shallow optic cup. Nasal disk margins hazy, temporal margins sharp. Vascular condition good, macular region and peripheral fundus clear. Pupil in the right eye (4.5 mm.) larger than pupil in the left eye (4 mm.). Good reaction to accommodation and to direct and consensual light. Fields and vision Figure 8.

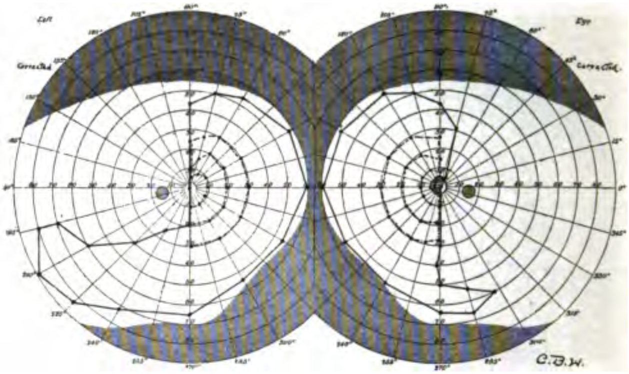

Fig. 8.—Case 5, chiasmal lesion. Hemiopic pupillary reaction well developed. Vision in left eye, 20/30—; in right eye, 20/200.

*Hemiopic Pupillary Reactions.*—With central fixation and rapid shutter movement the hemiopic pupillary reaction was most active in the lights turned into the quadrants of greatest field defect, measuring 7 on the right and 5 on the left by resistance and becoming equivocal with the pupillometer when the light areas were cut down to one-quarter the total area on the left, and one-fifth the total area on the right eye. With symmetrical eccentric fixation and rapid shutter motion, the reaction could be noted as far as 30 degrees above the fixation point along the line of cleavage of the blind and seeing field. In the left eye the upper and lower temporal quadrants showed a difference in reaction when tested in similar manner at 35 degrees from the center. By rotating a light concentric with the macula the reaction was readily noted in the upper fields.

Case 6 (Surgical No. 1135).—Acromegaly (incipient); hypophyseal struma. Nov. 26, 1913. Mr. H. A. E., aged 23. Frontal headaches for three months and failing vision for five months. Enlargement of hands first noticed two years ago. Prognathism began about one year ago. Other skeletal changes not marked. On admission found to be typical case of incipient acromegaly with hypophyseal struma.

*Eye Examination.*—Movements free, no nystagmus, von Graefe or limitations of movements. Fundi: optic disks pale but with a slight blush of injection present. Nasal side of disk paler than normal but still considerably better-colored than temporal side. Margins slightly blurred with a low-grade edema and some injection. Slight venous congestion. Fundi otherwise clear. Pupillary reaction to direct and indirect light and to accommodation very good. Fields and vision Figure 9.

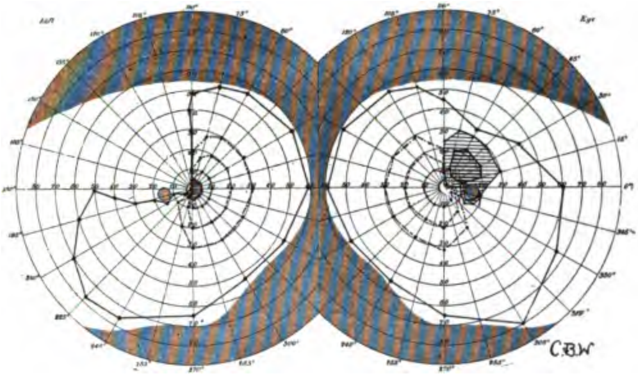

Fig. 9.—Case 6, chiasmal lesion. Hemiopic pupillary reaction present in the left eye, suggested in the right eye. Vision in left eye, 20/70+; in right eye, 20/30—.

*Hemiopic Pupillary Reaction.*—Present in the left eye on direct examination when the lights were rotated into the quadrant of greatest field defect, but there was a slight pupillary contraction as the light changed on to the blind retina followed by a dilatation to a larger size than noted at any time with the light on the seeing retina. This reaction measured 3 degrees on the resistance and was barely perceptible at full illumination with the pupillometer on the other eye. With central fixation the reaction could not be detected in any other quadrant. With the lights in the vertical meridian and fixation on a horizontal line so that the lights fell 20 to 30 degrees from the fixation point, in the temporal field, a little better reaction was obtained when the light was in the seeing field using a very rapid shutter motion to reduce asymmetry as much as possible. On the right eye the reaction was practically negative, although a suggestion of imbalance was present when the lights were in the axis of the greatest field defect.

These cases show the hemiopic pupillary reaction, varying in intensity with the form and degree of field defect, from a marked reaction in the case of the tract lesion to a rather early chiasmal lesion. The last case gives a fair idea of how much field defect may be present by this method without giving a definite difference in pupillary reaction.

In our series presented last year it was, in a way, unfortunate that no case was available in which the patient was hemianoptic in one eye and blind in the other eye. In such cases, when the consensual reaction to the blind eye is preserved, the pupillometer may be used on the blind eye to great advantage, since the pupil is not contracted thereby, and reactions even from weak lights are not masked.

CASE 7 (Surgical No. 1011).—Hypophyseal struma. March 10, 1914. Mr. A. G., aged 38. Failing vision began temporally in the right eye, progressing to complete blindness in the course of a year when failure of vision in the left eye began in the same manner. No diplopia, headaches, nausea or vomiting. Examination shows a rather typical case of hypopituitarism with struma.

*Eye Examination.*—No exophthalmos or nystagmus. There is some ptosis of the right lid and impaired convergence of the right eye possibly due to damage of the third nerve from a previous supra-orbital operation. Fundi: optic disk of the right eye shows a glistening pallor but lamina cribrosa stipplings considerably obscured by a thin layer of new tissue. Disk of the left eye has the same appearance, not quite so pale. Disk margins of each eye slightly blurred. Macular region, vessels and peripheral fundus in good condition. Pupil in the right eye (6.5 mm.) wider than the pupil in the left eye (5.25 mm.) with same illumination on both eyes of strength sufficient to give a clear view of the iris. Direct reaction of pupil in the right eye and consensual to pupil in the left eye good. Reaction to accommodation in the left eye slight. Fields and vision Figure 10.

*Hemiopic Pupillary Reactions.*—With central fixation the hemiopic pupillary reaction is most marked in the axis of least field defect (8 on the resistance) and less marked at 90 degrees to this axis, though still present. The equal areas could be shut down to a mere slit, or as far as symmetry could be maintained (about 1/3 cm. wide at the outer end) and with good dark adaptation the reaction could still be made out with the pupillometer on the other eye. Rotation of one light about the center gave the reaction as in the preceding cases, and rotating a small light on the perimeter, as described in the previous paper, a slight reaction on passing into the seeing field could be obtained as far from the center as 40 degrees. Using eccentric fixation with strong light from the rotary shutter a difference in reaction could be detected with rapid shutter motion as far out as 45 degrees. In all these tests fixation had to be aided by an enlargement of the fixation spot, so that while these measurements can only be considered as approximations, it must be said that they are very consistent.

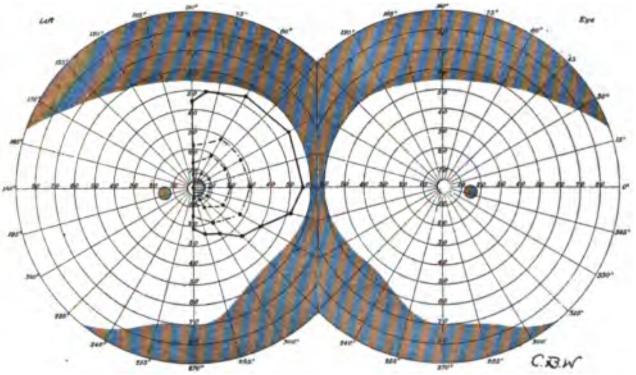

Fig. 10.—Case 7, chiasmal lesion. Hemiopic pupillary reaction marked. Vision in left eye, 20/200; in right eye, nil.

CASE 8 (Surgical No. 789).—Pituitary tumor. Jan. 11, 1914. Miss A. D. S., aged 41. Failing vision began about six months ago, progressing bitemporally to complete blindness in the left eye in a week or ten days. Headache, nausea and vomiting began five months ago but have been growing less severe on admission.

*Eye Examination.*—No exophthalmos or nystagmus; movements of eyes free and complete. Fundi show clear but not sharp disk margins. Optic cup moderate depth. Lamina cribrosa sharp. Disks are pale but not glistening. Pallor more marked in the left eye than in the right eye. Vessels, macula and peripheral fundus in good condition. Pupil in the right eye (4.5 mm.) smaller than the pupil in the left eye (6 mm.). Pupillary reaction in the right eye good with pupillary reaction in the left eye nil direct, but good consensually direct light, nil consensually. Reaction to accommodation not marked. Fields and vision Figure 11.

*Hemiopic Pupillary Reactions.*—The results in this case were very similar to the preceding one and fixation was very good. The hemiopic pupillary reaction with central fixation measured 7 on the resistance and could be seen with weak illumination with the pupillometer as in the preceding case. By concentric rotation tests and with eccentric fixation the reaction could be traced along the line of field cleavage to the region between 30 degrees from the center by the former, and 40 degrees by the latter tests.

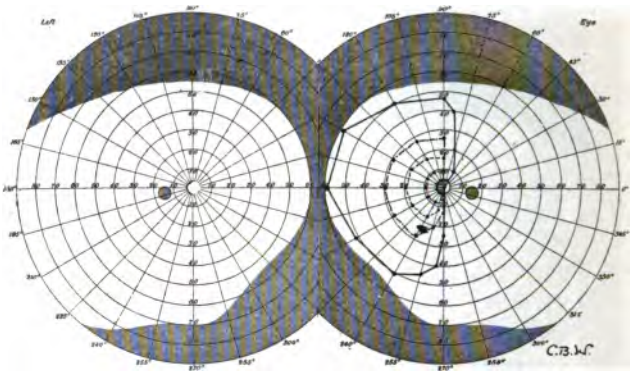

Fig. 11.—Case 8, chiasmal lesion. Hemiopic pupillary reaction well marked. Vision in left eye, nil; in right eye, 20/40—.

CASE 9 (Surgical No. 24).—Hypophyseal tumor typus Fröhlick. Feb. 25, 1913. Dr. R. E. S., aged 23. Vision began to fail three years ago, progressing to complete loss of vision in the left eye, one year ago, with temporal hemianopsia in the right eye. On admission patient found to be a typical case of hypopituitarism, typus Fröhlick.

*Eye Examination.*—No exophthalmos, diplopia or nystagmus. Both pupils slightly dilated, right (4.5 mm.) smaller than left (6 mm.) in moderate light. Fundus of Each Eye: Optic disk wide and very pale, especially in the left eye, with sharp outlines, shallow optic disk and clear lamina cribrosa. Pupil in the right eye (5 mm.) smaller than the pupil in the left eye (6 mm.). Good pupillary reaction to direct light in the right eye, no consensual from the left eye. No direct pupillary reaction in the left eye, but good consensual from the right eye. Fields and vision Figure 12.

*Hemiopic Pupillary Reactions.*—In spite of the small island of vision left in the temporal field the reactions were practically the same as in the preceding case, but not quite so strong. The hemiopic pupillary reaction measured 6 or a trifle over on the resistance board and the areas could be closed to a 0.5 cm. opening and still the reaction could be made out with the pupillometer. By concentric light and eccentric fixation tests with the rotary shutter the reaction could be followed as far out as 30 degrees from the fixation point. It could not be demonstrated by the use of small perimeter lights or by rotating the strong drum light, that the small temporal island of vision retained any pupillary activity. It is obvious that cases of this type are very valuable aids to the study of the extent of the pupillomotoric retina.

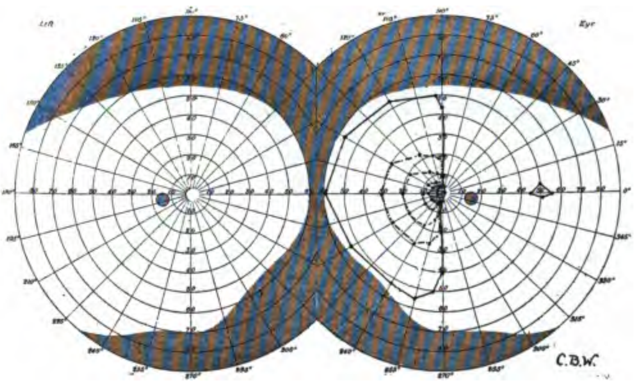

**Fig. 12.—Case 9, chiasmal lesion. Hemiopic pupillary reaction fairly marked. Vision in left eye, nil; in right eye, 20/100—.**

Case 10 (Surgical No. 578).—Hypophyseal struma. Nov. 13, 1913. Mr. W. L. M., aged 36. Vision in the left eye began to fail temporally two and one-half years ago and patient became blind in about one year. A few months later temporal hemianopsia began in the right eye. On admission patient was found to be in pituitary group with definite signs of hypopituitarism.

*Eye Examination.*—No ptosis; movements free and complete; both eyes rather prominent; no nystagmus; myopia in the right eye. Fundus of Each Eye: Disks very pale, especially right disk, which is glistening white. Margins sharp and clear. Optic cup wide and shallow with well-defined lamina cribrosa. Pupil in the right eye (4 mm.) smaller than the pupil in the left eye (5 mm.). Light thrown on the right eye gives a fair direct reaction and a good consensual reaction to the left eye. Direct and consensual reaction from the left eye nil. Fields and vision Figure 13.

*Hemiopic Pupillary Reactions.*—With central fixation and the light placed in the axis of greatest field defect there was, with rapid motion, only weak hemiopic pupillary reaction, becoming equivocal with two points of resistance. At best there was not much difference in size, but merely a pupillary bounce when the light was shifted to the seeing field. With the pupillometer the reaction was somewhat more distinct than the direct examination with full illumination, but it became rapidly weaker as the areas were shut down, until it disappeared when they were a little less than one-quarter size. Rotation of one light about the fixation point gave a weak hemiopic reaction in the upper temporal quadrant but eccentric fixation and other tests were not strong enough to be more than equivocal.

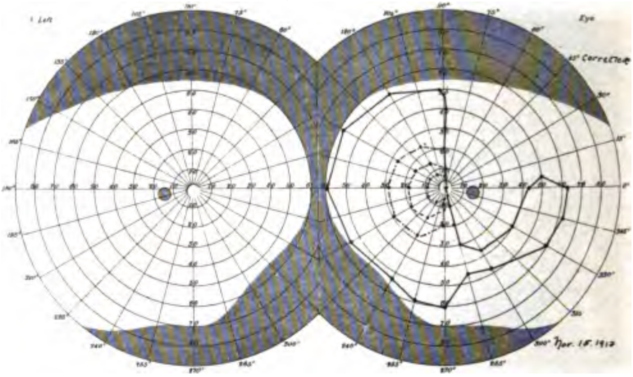

Fig. 13.—Case 10, chiasmal lesion. Hemiopic pupillary reaction present. Vision in left eye, nil; in right eye, 20/30.

In this case the hemiopic pupillary reaction was less than would be expected from the field defect as compared with other cases. In another case of complete bitemporal hemianopsia the pupils were even smaller and more sluggish in reaction than in this case, and no hemiopic pupillary reaction could be determined. In both these cases the pupils were smaller than usual with such a field defect and did not dilate as much as usual on complete dark adaptation. The range of pupillary motion seemed limited so that the rather spastic pupillary status apparently accounted for the weakness and failure of the reaction.

It seems then, that pupils which show the usual slight dilatation from their field defects and wide dilatation on dark adaptation have the most mobile reaction, and are most likely to give positive results with hemiopic pupillary tests. One of the best examples of the type of case will be reported first in proceeding with the remaining group of cases with posterior lesions.

Case 11 (Surgical No. 1049).—Right temporal lobe tumor, March 18, 1914. Mr. A. D., aged 26. Central vision in the right eye first noted to blur eleven months ago followed by slow failure of nasal field. In last few months temporary amaurosis has developed and continued with increasing frequency. Headaches, nausea and vomiting frequent but not as aggravating as usual with the fundus a picture of typical high grade choked disk. Occasional convulsions, uncertain gait, hallucinations and loss of memory.

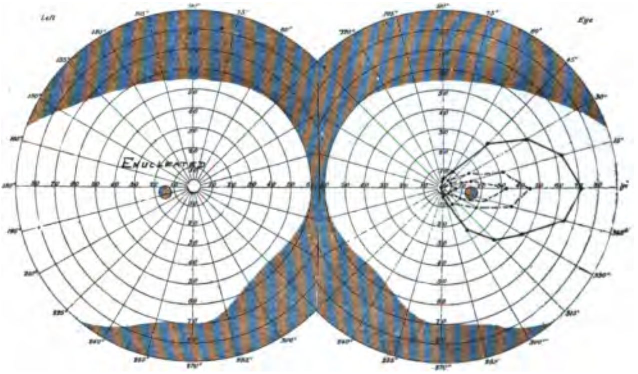

Fig. 14.—Case 11, right temporal lobe tumor. Hemiopic pupillary reaction marked. Vision in left eye, nil; in right eye, 20/20—.

*Eye Examination.*—Right Eye: Slight exophthalmos; no ptosis or nystagmus; movements free and complete. Typical choked disk in the right eye with a few punctate hemorrhages and exudate. Macular region peripheral. Fundus clear. Pupil moderately dilated (6.5 mm.) in ordinary light. Reaction to light and accommodation good. Fields and Vision Figure 14. Left eye enucleated thirteen years ago.

*Hemiopic Pupillary Reactions.*—The direct pupillary test with the rotary shutter showed perhaps the most striking and characteristic hemiopic pupillary reaction in the series. When the light was suddenly changed from the blind to the seeing field, there was a marked sudden contraction followed immediately by a slight dilatation. Equilibrium was rapidly reached with the pupil from 5 to 6 mm. in diameter. When the light was changed to the blind field no pupillary contraction or bounce was noted. The pupil slowly dilated from 7 to 8 mm. This cycle could be repeated *ad libitum* without weakening the reaction much, but when the patient was allowed to sit in the dark for ten minutes the reaction was noticeably strengthened. The reaction could be balanced when the temporal sight was decreased to the point 10. Rotation of one light about the center from the blind to the seeing retina gave the same phenomenon if the rotation was very rapid. If it was slow the pupillary jump on stimulating the seeing retina was very slight, or not noticeable and the same was true with very slow motion of the rotary shutter. With both lights in the seeing field equidistant from the fixation point pupillary response was of the same character as seen in the normal eye. Tests of the reaction along the line of cleavage between seeing and blind fields could be made by tilting the lights into an axis of 45 degrees or 135 degrees and using eccentric fixation. Then the reaction could be traced out to 25 degrees from the fixation point where it became equivocal.

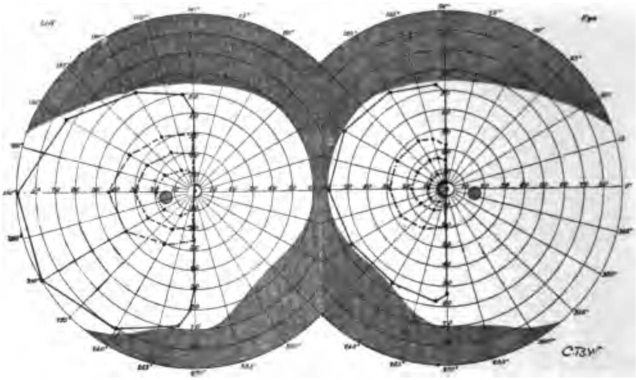

Fig. 15.—Case 12, left posterior lobe lesion. Hemiopic pupillary reaction marked. Vision in left eye, 20/30; in right eye, 20/200.

CASE 12 (Hospital No. 1046).—Left posterior temporal lobe lesion. Nov. 29, 1913.—Mr. L. A. C., aged 22. Headache, weakness and numbness of right arm and leg increasing for two months. On examination patient was found to have a right homonymous hemianopsia, motor aphasia, left paralysis and paresthesia, headaches, choked disk, etc., leading to diagnosis of a left posterior lesion. At operation three echinococcus cysts about the size of walnuts were removed from posterior part of left hemisphere.

*Eye Examination.*—No exophthalmos, nystagmus, diplopia or muscular imbalance made out. Fundus of each eye: typical choked disc, 3 D on the right and 4 D on the left. Pupil in the right eye (5.5 mm. in diameter) larger than the pupil in the left eye (4.5 mm.) with good reaction to light and accommodation.

*Hemiopic Pupillary Reaction.*—Marked in both eyes, neutralizing when the light in the seeing field was weakened to point 8 in the right eye and point 7 in the left eye. When the light was changed to the blind field there was no reaction, simply a slow dilatation. Moving the fixation point up or down in a vertical line the hemiopic pupillary reaction could still be made out at 40 degrees. With both lights equidistant from the center and in the seeing field the pupillary phenomena were normal. Rotation of one light rapidly about the center gave a similar reaction to the rapid shutter, but not quite so strong. Slow rotation gave a similar reaction to the slow shutter change. With the pupillometer the consensual hemiopic reaction disappeared when the openings were about five-sixths closed, but at best considerably less marked than with direct examination.

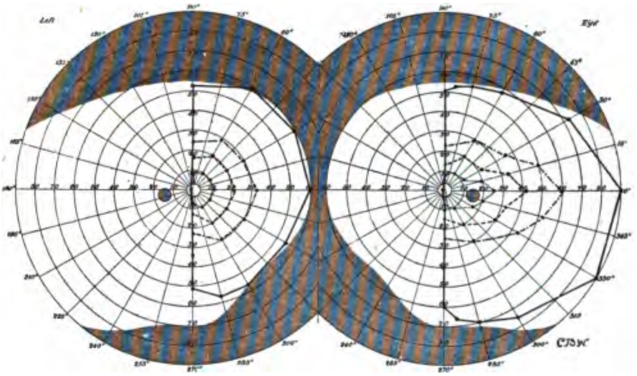

**Fig. 16.—Case 13, right parietal lobe tumor. Hemiopic vision in left eye, 20/20; in right eye, 20/20. Pupillary reaction well marked.**

CASE 13 (Surgical No. 397).—Right parietal lobe tumor. Sept. 15, 1913.—Mrs. C. R. C., aged 44.—About eight months ago left homonymous hemianopsia developed and escaped detection until complete. Later headaches. Choked disk (3½D), anesthesia and weakness of left side of body, dizziness, Babinski, etc., led to diagnosis of right occipital lobe tumor with cerebellar neighborhood signs. Right subtemporal decompression June 10, 1913. On Sept. 21, 1913, a large tumor was successfully removed from the right parietal lobe by Dr. Cushing.

*Eye Examination.*—Both eyes: No exophthalmos; movements free and complete; no nystagmus; media clear. Fundi show typical picture of receded choked disks of low grade after decompression. With no central scotoma and good macular vision, the left pupil (5 mm. diameter) was definitely larger than the right pupil (4 mm.) in ordinary illumination. Good reaction to direct and consensual light, slight reaction to accommodation. Fields and vision Figure 16.

*Hemiopic Pupillary Reactions.*—With the lights in horizontal position and fixation in the center the hemiopic pupillary reaction was present in both eyes, slightly more marked in the left eye and balancing when the light in the seeing field was decreased to the point 4½. If fixation was made above or below the center equidistant from the two areas the reaction became less noticeable until it could not be made out at about 30 degrees from the center. When the light was changed from the seeing field to the blind field there was occasionally noted a slight pupillary bounce followed by a dilatation to a larger size than noted with the light in the seeing field. With both lights equidistant from the center in the seeing field the reaction was the same as in the normal eye. Using the pupillometer the hemiopic character of the reaction in the small pupils could barely be made out with any illumination, and would pass as negative without other evidence.

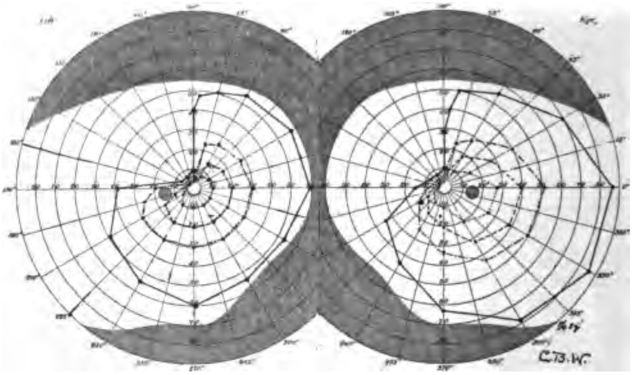

Fig. 17.—Case 14, temporal lobe tumor. Hemiopic pupillary reaction present. Vision in left eye, 20/30; in right eye, 20/30—.

CASE 14 (Surgical No. 968).—Temporal lobe tumor. Feb. 27, 1914.—Mrs. E. A. J., aged 43. Diplopia and occasional headaches for over six months. Slow progressive tendency to left homonymous hemianopsia beginning in upper quadrants. On operation, right subtemporal decompression, about 50 c.c. of what was apparently cystic fluid was withdrawn by needle from the second temporal convolution, leaving the temporal lobe collapsed.

*Eye Examination.*—Slight exophthalmos in both eyes, possibly greater on right. Considerable weakness of right external rectus. Fundus of each eye: increase in venous engorgement and tortuosity with tendency to partial embedding at disk margins nasally. Margins, especially nasally, are edematous and elevated 1½ D. Picture, if anything, more aggravated on right. Pupils equal (5 mm.), react well to light, both directly and consensually. Reaction to accommodation present but not marked. Fields and vision Figure 17.

*Hemiopic Pupillary Reaction.*—When the lights were rotated into the axis of greatest field defect there was a noticeable difference in pupillary reaction which could be balanced by weakening the light in the blind field to the point 3. There was a very slight contraction when the light changed to the seeing field. When the lights were in the horizontal position or at 90 degree to the former position, however, the reactions were so nearly normal as to be equivocal. Other tests were negative.

This case is interesting as a posterior lesion in comparison with Case 6 as an anterior case, in giving some idea as to how early a difference in pupillary reaction between the blind and the seeing retina may be detected with relatively strong lights on direct examination.[7]

## SUMMARY

From a study of these cases it would appear that the idea of using relatively strong symmetrical illumination has not been an unfruitful method of examination for the hemiopic pupillary reaction. In fact, it has been rather disappointingly fruitful from a standpoint of clinical diagnosis, since apparently a very similar type of reaction has been found in cases of both anterior and posterior lesions.

In the most characteristic form of the reaction, there is, with central fixation on the rotary shutter, no pupillary bounce whatever, as light is changed from the seeing to the blind field, with any rate or interval of shutter movement. The pupil simply dilates slowly in the course of two or three seconds, being somewhat more suggestive of a pupil being released from the effects of light adaptation, than of an ordinary pupillary dilatation as usually seen in a light reaction test. On changing back to the seeing field there are two kinds of reaction according to the rate of shutter movement. If the change is slow so that light adaptation may have time to take place to a certain extent, the pupil will slowly contract to a certain size. If, however, the shutter movement is very rapid, the pupil contracts with a sudden jerk to a size smaller than with slow movement, and it dilates as light adaptation comes on, to a size about the same as that with the slow shutter movement. This effect of light adaptation varies with the strength of illumination used, being much more marked with the strong illumination used in direct examination than with the weaker illuminations which may be used in consensual examinations.

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[7] During the interval between going to press and the proof reading, two more posterior cases have been examined and have strengthened our conclusions. One was a case (Surgical No. 1114) of cerebral embolus and the other (Surgical No. 1148) was a temporal lobe cyst opened and drained as in Case 14. Both had practically complete homonymous heminopsia of between three and four months duration and both showed a well marked hemiopic pupillary reaction.

In addition to this characteristic type of reaction there are those cases in which there is only a slight pupillary bounce when the light changes to the seeing field, but the pupil is noticeably larger with the light in the blind field than when in the seeing field. Finally, as the reaction becomes equivocal, it is noted that without distinct difference in pupillary size at rest, there is a more marked pupillary bounce on one side than on the other.

These reactions in their various stages have been found in anterior lesions as well as in posterior lesions. Indeed, one of the most striking examples was in a posterior case. The phenomenon has been found in rather early stages of incomplete hemianopsia of both bitemporal and homonymous types. It has been found in tract and chiasmal lesions both with and without relative central scotomata.

The topical diagnosis in the cases presented, is without question in the anterior group of chiasmal and tract lesions. In the group of posterior cases the diagnosis is almost as certain, though without necropsy it would not be possible to say absolutely that there was no involvement of the pupillary tract, or of the primary ganglion centers. In addition to this rather slight ray of hope, there is the possibility in some of the cases that secondary degeneration may have affected the pupillometer tract as has been suggested by Rothman. In this connection it would be extremely interesting to follow for from six to eighteen months the pupillary reactions in a very early case of hemianopsia due to capsular apoplexy.

Previously we have been contending largely with the problem of dispersion light, but now, having disposed of that problem more or less satisfactorily, we are evidently confronted by a still more serious complication in the retinal properties of light and dark adaptation. One can blind one half of the retina by looking at the edge of a large bright surface and obtain a difference in pupillary reaction in the normal eye with the rotary shutter. Indeed, the action of the normal eye with direct test on the rotary shutter shows the complication of light adaptation. In complete homonymous cases of whatever origin, Cushing has long noted a large pupil in the eye having lost the temporal field, that is, having lost the greater area of active retina. I have seen this phenomenon several times and three such cases are given in this report. Two of them are posterior lesions. This pupillary status appears too constantly for a sympathetic phenomenon and it cannot be explained by assuming all the pupillomotor activity to be resident in a small macular area, nor does it agree with the idea that the reflex pupillary arc is still intact on the blind side in posterior lesions, even if it were known that the pupillomotoric area were quite large and extended farther on the nasal retina. It may be more easily understood when we consider the light and dark adaptative effect of the two unequal retinal areas on their respective pupils.

From the examination of these cases it would appear that the blind retina has, to say the least, either lost or decreased in light and dark adaptive effects on the pupil. If light and dark adaptative pupillary reactions exist merely as a regulating mechanism, and involve the presence of retinal pupillomotor activity, then we are forced to the conclusion that the retina must either be weakly pupillomotoric to a considerable distance from the macula (30 to 40 degrees), or it is pupillomotoric in a small central area which is sufficiently active, when split, to give a difference in reaction to that distance by asymmetry. In either case, the pupillomotoric activity must fail in the blind retina in order to give the results obtained in these examinations. This has occurred, apparently, in posterior cases against rule (obtained from certain rare cases of subjective blindness from posterior lesions with retained pupillary reactions) that hemianoptic posterior lesions do not disturb the pupillomotoric activity of the blind retina. Possibly the rarity of these cases offers an explanation, that is, that the percentage of cases in which the hemiopic retina is left intact pupillomotorically is no greater than in cases of subjective blindness from posterior lesions, where pupillary reaction is retained. However that may be, it seems reasonably certain that the light adaptive properties are sufficiently damaged in posterior cases of hemianopsia to give a pupillary reaction closely simulating the hemiopic reaction of the pupillary reaction of anterior cases.

Aside from these difficulties, the possible complication of psychic reactions must also be considered. The concentric movement reflex which I have previously mentioned, and could demonstrate in many of these cases, is doubtless simply a light adaptation phenomenon from which the psychic element may be worn away by repetition. The precautions taken before proceeding to the examination aid considerably in eliminating psychic reactions. But the consistency with which the hemiopic pupillary reaction occurs and may be repeated, argues much against psychic complications of any importance. With good cases which could stand the strain of fixation for a considerable period, I have demonstrated the reaction to chiefs and colleagues time after time, as long as they cared to look at it, and none had difficulty in making it out with the unaided eye.

Indeed, in the most marked cases the reactions are so striking that with rough methods one would expect to find a semblance of the phenomenon, and such has been found to be the case. With a pocket flash-light or an electric ophthalmoscope sufficiently strong to give a good view of the pupil, one can get a distinct though not a marked difference in pupillary reaction whenever, on repeated trials, the light is flashed from roughly symmetrical positions on each side of the fixation point. If two flash-lights are held in symmetrical positions and flashed alternately, the reaction is still better but never as marked, or as constant as with the rotary shutter. The fact that the reaction can be detected in marked cases by rough methods both in the anterior and in the posterior cases has an interesting significance in considering the great variety of results reported by observers using these methods.

If the retina has a low grade of pupillomotoric activity or a combination of light adaptive and pupillomotoric properties to some distance from the macula, as my results seem to show, how may the work of Hess be explained? Hess threw a small light on his own optic disk and obtained no reaction by the entopic method when he moved the light above or below concentric with the macula, on to the seeing retina. In fact, sometimes he obtained a better reaction when he passed from the retina on to the bright blind spot because of more reflected light. It is obvious that if the retina about the blind spot had a pupillomotor sensitiveness practically equal to the stimulation of the macula by reflection from the blind spot itself, then the results of Hess could be obtained in the same manner but for an entirely different reason than he supposed, namely, that the peripheral retina had no pupillomotoric activity. Accordingly the size, shape and illumination of the equal areas of the rotary shutter were designed to get the maximum effect from as large an area of retina as possible, and still keep the dispersion light reaching the macular region constant. With this instrument the reflection from the blind spot simply balances its lack of pupillomotor activity, so that its asymmetrical location apparently has no effect.

Altogether from a careful study of the cases reported herein, apparently many conclusions may be drawn, of which the following would appear particularly justifiable.

## CONCLUSIONS

1. A weak hemiopic pupillary reaction may be masked by the pupillometer light when observed consensually.

2. Light and dark adaptive phenomena, in addition to dispersion light, seriously complicate the hemiopic pupillary reaction.

3. The hemiopic pupillary reaction is definitely present in anterior lesions, when examined by the rotary shutter method.

4. The hemiopic pupillary reaction is also present in cases having every clinical evidence of being purely posterior cases, although autopsy examination is

necessary to prove absolutely that there is no involvement of the optic tract or primary ganglion centers.

5. Although it may be concluded from the examination of these cases that the peripheral retina does possess a weak pupilomotor sensitiveness, there is no evidence that the hemiopic pupillary reaction has any topical diagnostic value.

I am deeply indebted to Prof. Harvey Cushing not only for kindly facilitating this investigation but also for generously allowing me the privilege of presenting this particular phase of his cases.

233 Charles Street.

# MEMORANDA

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# EXPERIMENTAL RESEARCHES IN METHYL ALCOHOL INHALATION *

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Notwithstanding the earnest efforts of the ophthalmologists and the various committees for the prevention of blindness, to disseminate the knowledge of the poisonous qualities and effects of methyl alcohol, it is still a lamentable fact that many members of the medical and legal professions and the vast majority of the laymen, especially the workers using it, are either ignorant of or do not acknowledge the truth. The effect on the eyes and general health from drinking wood alcohol has been proved experimentally in animals, and has been corroborated in clinical practice; but the effect from its inhalation alone on the eyes has never heretofore been demonstrated experimentally. Although considerable clinical data has been collated tending to show an effect similar to the one obtained from its imbibition, many doubt and some dispute its power to affect the human organism deleteriously through inhalation alone. To prove this fact, coupled with a desire in the interest of humanity to lessen the number of the blind and to conserve life, by advocating proper preventive or precautionary measures for workers using it, induced us to undertake these experiments.

Before giving them in detail, it may be of interest briefly to refer to the importance of the subject to the body politic.

We have found that there have been about one hundred cases of amblyopia and death from inhalation

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

of wood alcohol recorded in the literature to 1912, and we are of the opinion that this constitutes a very small percentage of the total number of cases which have actually occurred, as a great many either have not been recorded, or have gone unrecognized. Of those that have appeared in the literature, the question whether inhalation alone produced the condition, has usually arisen. In conducting our investigations, we have endeavored to exclude all possibility of the entrance of the wood alcohol into the system except by inhalation, and we feel sure that we have succeeded in producing amblyopia and death in that manner, as we contemplated at the beginning of these researches.

In 1880 about 400,000 gallons of wood alcohol were manufactured, while in 1910 the amount had increased to over 8,000,000 gallons; consequently it appears that the industry has become firmly established. That being true, it is for the chemists and medical profession to so arrange for its use or composition, that while it may serve its purpose in the arts and industries, it shall not levy too heavy a toll on the artisans, and indirectly on the state by causing blindness or death. Its use is extensive, about 2,000,000 people working in trades are using it in the United States alone, and as the foregoing figures indicate, is increasing annually.

According to the 1904 United States Census, the following industries and trades were using wood alcohol.

| | |
|---|---|
| Painters, glaziers and varnishers... | 278,000 workers |
| Launderers ........................ | 386,000 workers |
| Boot and shoemakers.............. | 208,000 workers |
| Barbers ........................... | 131,116 workers |
| Printers and lithographers......... | 155,000 workers |
| Woodworkers—piano, furniture, etc. | 112,000 workers |

About 500,000 workers divided among dye-work operators, brass-workers, cabinet-makers, gold and silver workers, harness-makers, hat-makers, leather-workers and tanners, metal-workers, photographers, book-binders, chemists, etc.

The subject is one of increasing importance on account of the new employers' liability law in New York and other states. At present it is not even mentioned specifically as a hazard, but when the importance of the effect of its inhalation is realized, cognizance of this omission must surely follow.

The manufacturers claim that even admitting that commercial wood alcohol is poisonous, the refined product known as Columbian Spirits is not. We have proved conclusively that the refined spirit is equally as dangerous as the unrefined; and indirectly more likely to destroy vision and life on account of the absence of the disagreeable odor to warn one of its presence.

Our original intention was to note the effect of methyl alcohol inhalation in chronic cases, that is, those which had been exposed to its influence in small quantities over a long period. But the time allowed in which to prepare our paper for the meeting was most inadequate to conduct our investigations along these lines, so we were obliged to confine our attention principally to the more acute phases of the matter under consideration.

The apparatus used in our investigations consisted of a wooden box which was 32 cm. wide, 47 cm. long and 63 cm. deep (high) with an opening on each end about 1 cm. in diameter. In one end a mica valve was very delicately adjusted, allowing air to pass out. At the other end was an attachment for tubing which could be connected with an air-tank, or copper coil, and which acted as an inlet for fresh air. The top, in which were two square glass windows, could be fastened down by eight thumb-screws. The temperature of the air in the box for the experiments averaged about 68 F., while the hygrometric readings indicated an average of about 60 per cent., thus to a certain extent constituting conditions similar to those in which some of the workers with methyl alcohol use it, for example, varnishers in beer-vats, etc.

The wood alcohol used in the experiments consisted of both the commercial and the Columbian Spirits, and they were obtained from Eimer and Amend, chemists of New York City.

## RESEARCHES IN METHYL ALCOHOLIC INHALATION IN ANIMALS

Experiment 1.—Acute poisoning in dog. Dec. 3, 1913: A dog, male, weight 4 kg, 700 gm. (about 10 pounds), fed one hour before experiment. At 4:15 p. m. was placed in the box with 2 ounces of wood alcohol on a sponge, inlet and outlet of box open for fifteen minutes during which time dog jumped and barked, then openings were closed. Room temperature 64 F. Interior of box temperature 70 F. Dog at 4:50 p. m. was lying quietly on his side. At 5:30 p. m. box was opened, dog appeared intoxicated; fell on attempt to walk. Moved his head from side to side.

December 5: Breakfast: meat and bread, at 11:30 a. m. At 4:30 p. m. dog was placed in box with 4 ounces wood alcohol on two sponges and box closed. At 4:40 p. m. dog was lying down, abdomen up, breathing heavily. At 4:50 p. m. dog's mouth was slightly open, tongue projecting and body in a position of opisthotonos, abdomen upward, animal moving fore feet feebly. At 5:05 p. m. dog unconscious, respiration 50 per minute. At 5:25 p. m. box opened; dog taken out apparently dying.

December 8: Dog recovered in thirty-six hours, and whereas on the 5th he was dull, he now appeared lively and well. Was placed in box with 5 ounces wood alcohol; box closed at 5:10 p. m. At 5:50 p. m. dog was taken out intoxicated, breathing heavily.

December 9: Dog appeared lively and well, and remained so.

December 30: Dog was placed in box with 5 ounces wood alcohol at 1:50 p. m. At 5:10 p. m. was observed lying on right side with tongue protruding and twitching of fore legs; was taken out of box. Examination: Temperature 90 F.; eye tension, Schiötz tonometer 26 mm. Hg, although its palpation seemed much lower, tonometer reading possibly little higher due to pressure of eyelids and to contraction of lid muscles; respiration 29 per minute, labored; blood-serum not alkaline to phenolphthalein; slightly alkaline to litmus; aqueous humor not alkaline to phenolphthalein; slightly alkaline to litmus; twitching of eyelids; cornea only slightly hazy; pupils 3 mm., no light reaction; fundus of eye, optic disk congested with edema; veins dark and dilated, arteries small.

December 31: Dog apparently dying; respiration 10.

Jan. 2, 1914: Dog found dead in morning.

*Remarks.*—This dog had four exposures to wood alcohol inhalation, spread over a period of one month. He died after the fourth exposure. It is interesting to note the continuous movements of the head from side to side, which we venture to attribute to an irritation of the semicircular canals of the internal ear.

EXPERIMENT 2.—Acute poisoning in guinea-pig. Dec. 5, 1913: Bell jar used, 28 cm. in diameter, 18 cm. high. A guinea-pig was placed under jar with 2 ounces wood alcohol. At 4:45 p. m. animal was quiet; eyes half closed. At 5:05 p. m. respiration 84 per minute, labored; entire body constantly moving; convulsive type, moves as if intoxicated. At 5:25 p. m. intoxicated—saved for next experiment.

December 9: Guinea-pig again placed under jar with 4 ounces wood alcohol at 4:55 p. m. At 5:20 p. m. gait uncertain; falls on side. At 5:28 p. m. hind legs week. Respiration 105 per minute. At 5:48 p. m. animal lying down breathing regularly though unconscious.

December 10: Guinea-pig found dead; head was severed and preserved in formaldehyd solution.

*Remarks.*—The guinea-pig became intoxicated after the first and unconscious after the second exposure. The convulsive type of respiration is interesting, because it shows that the poison acted on the respiratory center. A control guinea-pig was kept under jar without methyl alcohol vapor for same length of time, and remained normal while under observation.

Experiment 3.—Dec. 9, 1913: Two guinea-pigs—weight, Pig 1, 300 gm., Pig 2, 295 gm., were placed under bell jar with 10 ounces wood alcohol, at 4:25 p. m. At 5:25 p. m. both were intoxicated and semicomatose and were taken out.

December 11: Both pigs were found dead on the morning of December 11, having lived about thirty-six hours. Post mortem of Pig 1 showed large intestines bloated; mucous membrane of stomach showed rough and reddish spots about 2 cm. in diameter; kidneys dark red; suprarenals pale yellow; no hemorrhage; bladder and gall-bladder full; heart full of reddish-black clots. Head and spine preserved in formaldehyd solution.

*Remarks.*—Both guinea-pigs died within thirty-six hours from time of inhalation of wood alcohol, exhibiting characteristic changes in the stomach.

Experiment 4.—Dec. 11, 1913: A guinea-pig, weight 500 gm., was placed under jar with 2 ounces of Columbian Spirits, at 4 p. m., and taken out at 5 p. m. Respiration 70 per minute, dyspneic, labored; occasionally about every ten or fifteen seconds body spasmodically contracted in the anteroposterior direction; hind legs weak.

December 13: Guinea-pig lively. Placed under jar with 2 ounces of Columbian Spirits at 4:03 p. m.; at 4:06 p. m. rubs nose. At 4:10 p. m. respirations 55 per minute; 4:50 p. m. convulsive lateral movements of head. Hygrometer, 70 in jar at end of one hour. At 5:05 p. m. slight weakness in hind legs; when taken out, weight was 450 gm.

December 16: Pig dead; post mortem: stomach, multiple hemorrhages; heart contains clots; suprarenal glands dark red; medullary portion dark red; hemorrhage in right suprarenal.

*Remarks.* — The guinea-pig which received the Columbian Spirits inhalations became intoxicated after each exposure and exhibited convulsive movements of the head and body. The lateral movements of the head similar to those noticed in dog (Experiment 1) are worthy of mention. A control guinea-pig remained normal while under observation.

Experiment 5.—Effect of methyl alcohol inhalations, with and without ventilation. Dec. 18, 1913, at 4:05 p. m., a dog, weight 3,980 gm. (about 9 pounds), lively, eyes normal, was placed in box with inlet and outlet working. Through inlet air is pumped in at rate of 28 pumpings per minute under 2 pounds pressure. The air passes through a Woulfe bottle, which contains 3 ounces of Columbian Spirits. The vapor is thus forced through a copper spiral or coil placed in a pail of water, under which is a Bunsen burner to heat water and coil. After passing through coil it enters box warmed. At 5:05 p. m. dog was taken out of box lively and apparently unaffected.

December 20: Dog placed in box for twelve minutes. Experiment as before, allowing vapor to pass directly from Woulfe bottle into box without being warmed. Air-pressure 2½ pounds—26 pumpings per minute. At 5 p. m. hygrometer in box, 40. After remaining in box 5⅔ hours dog was lively, showing no apparent effect of vapor.

December 22: Condition as before. Dog in box from 12 m. to 3:30 p. m. having had 5 ounces of Columbian Spirits pumped in with no apparent effect.

December 27: Placed in box at 11:55 a. m. with 2½ ounces wood alcohol pumped in under 2½ pounds pressure; taken out at 5 p. m. lively; no apparent effect.

December 29: Through error of attendant, dog placed in box with 5 ounces wood alcohol without free ventilation. Examination at 4 p. m. found dog lying on right side, with tongue protruding, frothing at mouth, hair moist; was taken out of box. Respirations 20 per minute; deep reflexes absent; unable to stand; comatose.

December 30: Dog still comatose, lying on right side. Respiration shallow, irregular 50 per minute. Temperature 90. Eyes: tension from 6 to 8 mm. in both corneae only faintly hazy; sensibility slightly diminished; pupils 3 mm. light reaction; aqueous humor not alkaline to phenolphthalein, neutral to litmus; fundus, optic disks red and edematous; arteries small, veins dark red and dilated. Blood-serum not alkaline to phenolphthalein, slightly alkaline to litmus.

December 31: Dog found dead in morning by attendant.

*Remarks.*—This experiment is a striking illustration showing the diminished resistance to the inhalation of Columbian Spirits when a very free ventilation was abolished. Moreover, we believe that the repeated exposures to the inhalation of Columbian Spirits (even with free ventilation) did have some effect on the dog, and that the last exposure had a quick fatal effect on account of the previous exposures.

EXPERIMENT 6.—Acute poisoning in rabbit. Dec. 13, 1913, a rabbit, weight 1,600 gm. (3½ pounds), was placed under a large bell jar with 5 ounces Columbian Spirits, and three minutes later was rubbing its nose. Respirations 50 per minute. Every seven or eight respirations spasmodic contraction of diaphragm resembling hiccup. At 4:55 p. m. respirations 32 per minute, with occasional convulsive rapid shortening of the body. Hygrometer 80. Taken from under jar at 5:05 p. m.

December 20: At 4:45 p. m. animal was placed under large jar with 5 ounces Columbian Spirits. At 5:45 p. m. respirations 40 per minute; hygrometer 70; taken out from under jar, not much effect shown.

December 23: At 3:40 p. m. animal under jar with 3 ounces Columbian Spirits. At 4:45 p. m. respirations 38; hygrometer 70; temperature in jar 68. Taken out in good condition.

December 27: At 11:55 a. m. animal under jar with 5 ounces Columbian Spirits. At 12:55 p. m. hygrometer 55; at 1:35 p. m. respirations 57. At 2:30 p. m. respirations 50. At 4 p. m. animal lying on side quietly; breathes regularly and deeply, 22 per minute. At 4:40 p. m. respirations 16; at 4:45 p. m. respirations 4. Taken out from under jar; cornea hazy; no lid reflex; no light reflex; nose and ears cold. Temperature by rectum 90 F.

December 29: Found dead in morning. Eyes preserved in formaldehyd solution.

*Remarks.*—The rabbit appeared to be more resistant to the Columbian Spirits than the dog was. Coma and death came only after the fourth exposure, although relatively larger amounts of Columbian Spirits were used. The same spasmodic contractions of the body and diaphragm were noticed as in former experiments.

EXPERIMENT 7.—Result of subcutaneous injection of Columbian Spirits. Dec. 20, 1913, a healthy, active, female guinea-pig, weight 375 gm., was given a subcutaneous injection of 5 c.c. of Columbian Spirits at 5:07 p. m. Almost immediately animal became quiet and fell in coma. Respirations 108. At 5:10 p. m. occasional spasms occurred; respirations 62. Thirty seconds later respirations 48, with slight spasm; at 5:11 respirations 48; at 5:14 p. m. respirations 44; at 5:35 p. m. respirations 44, irregular and spasmodic. At 5:45 p. m. cornea reflex intact; animal lies in profound coma; respirations 34; occasional short convulsive movement which became general. At 6 p. m. respirations apparently stopped, while heart continues to beat and convulsive movements of head continue. Pulse 96; animal dying in convulsions. At 6:15 p. m. animal dead. Eyes enucleated placed in formaldehyd solution.

*Remarks.*—This experiment was performed with the idea of watching the general symptoms in a guinea-pig produced by a rapid action of the refined wood alcohol (Columbian Spirits). The almost instantaneous coma, followed by convulsions, the continuation of the heart-action long after the cessation of the respiration, and most curiously the disappearance of all reflexes long before the corneal reflex, deserve special mention.

EXPERIMENT 8.—Acute poisoning in dog. Jan. 3, 1914, a dog, male, weight 5½ kg. (12 pounds), was placed in box at 12:55 p. m., inlet and outlet closed; 5 ounces methyl alcohol commercial. At 1:50 p. m. animal staggered about, frothing at mouth, and at 2:50 p. m. was unable to stand; at 4:10 p. m. lying on right side; respirations 25, deep and labored. Dyspnea at 4:55 p. m.; respirations 24; pulse 150; eyes, cornea clear, sensibility diminished; pupils 2 mm. contracted; no light reaction; fundus, optic disks hyperemic, with edema; arteries small, veins dark, slightly dilated. Tension, left 18.5. Aqueous humor slightly alkaline to litmus, not alkaline to phenolphthalein. Respirations short; inspiration prolonged, expiration about as one to four. Hygrometer 55; temperature in box 66 F. Dog remained in comatose condition from January 3 to 5; was found dead the morning of the 5th, lying in same position as when examined on January 3 and 4.

*Remarks.*—Death was produced by one prolonged exposure (four hours). The intra-ocular pressure was not low January 3, soon after the exposure, and the animal lived more than twenty-four hours, a fact which confirms previous conclusions, namely, that when the tonometer shows a normal intra-ocular pressure, the animal, though in an apparently dying condition, will live at least twenty-four hours or more, and an intra-ocular pressure below 5 mm. Hg is a sign that death will follow within twenty-four hours.

Experiment 9.—Effect of repeated methyl alcohol inhalations of short duration extending over a period of seven weeks. Jan. 6, 1914, a male dog, weight 4,900 gm. (about 11 pounds), was placed in box at 1:30 p. m. with 5 ounces methyl alcohol. Box closed; no ventilation. At 4 p. m. dog was standing; at 4:30 p. m. taken out; respirations, 35; expiration prolonged, animal lying on left side frothing at mouth. Eyes, tension 26; pupils widely dilated, size 6.7 mm.; no light reaction; fundus, veins dark and full, arteries small and tortuous; color of disks normal, slight congestion at the superior nasal quadrant.

January 7: At 9 a. m. the dog still sluggish and inactive; temperature, 100 F.; was placed in box under conditions similar to the foregoing and at 12 m. was taken out. At 1 p. m. animal was intoxicated; at 2 p. m. was shivering and weak in hind legs, and unable to control them perfectly. Once he attempted to move forward with his front feet making no attempt to move the hind legs until the belly reached the floor, when he fell over on his side and then pulled up his hind legs. Pupils contracted to 3 mm.

January 8: At 3 p. m. respirations 18; pulse 145; dog active. Placed in box; similar condition as above. At 4:35 p. m. animal taken out; respirations 20; pulse 120; temperature 100 F.; eyes, pupils wide, 5.6 mm., reacting sluggishly to light; cornea clear, reflex slightly diminished; fundus, veins dark, almost black, disks edematous; physiologic excavation less deep; a small, exceedingly bright red spot appears to temporal side of disk, right eye; hygrometer 50; temperature in box 60. At 4:53 p. m. pilocarpin 2 per cent. solution in O. D.; physostigmin (Eserin) 1 per cent. solution in O. S. At 5:14 p. m. pupils same size, from 5 to 6 mm., not affected by the use of miotics.

January 10: In box with 5 ounces methyl alcohol; box closed for 1¼ hours; dog lively. Cornea only very slightly hazy, pupils from 5 to 6 mm., no light reaction. Temperature 100 F.; pulse 146; respirations 20; prolonged expiration; shivers as if cold. Fundus veins dark, cornea sensibility diminished. At 4:45 p. m. pilocarpin 2 per cent. solution dropped twice in O. D. At 5:15 p. m. pupils contracted to 3 mm.; pupils react well to light, but pupil in O. S. does not contract to size of pupil in O. D.

January 12: Dog in box one hour; was taken out active. Pulse 160, respirations 16.

January 13: Respirations 28, pulse 100. At 4 p. m. in box with 5 ounces methyl alcohol. Taken out at 5:20 p. m. Hygrometer 55; temperature 59 in box. Dog temperature

101. Cornea slightly hazy, no light reaction; optic disks paler than in normal dog examined.

January 14: 2:30 p. m. temperature 102, respirations 18; animal placed in box. At 3:30 p. m. taken out of box. Temperature 100; respirations 16; animal fed at 4:30; good appetite.

January 15: In box one hour before experiment; temperature 100, respirations 15; after experiment, temperature 101, respirations 15.

January 17: In box one and one-half hours; temperature 102, respirations 18 before experiment; temperature 100, respirations 18 after experiment.

January 22: In box one hour, every day except January 20, under conditions same as beginning. Fundi less pale than few days ago; vessels dark.

January 23, a. m.: In box one hour; temperature before 101; after 100.6. January 23, p. m.: In box one hour; temperature before, 101; after, 102.

January 24, morning: Temperature before entering box 101; one hour after, 100.

January 24, afternoon: Temperature before entering box 102; one hour after, 102.2.

January 26: Animal in box at 2:45 p. m., temperature 102.8; out at 3:45 p. m., temperature 102.

January 27: Animal in box at 8:40 a. m., temperature 102.2; out at 9:40 a. m., temperature 100.6.

January 27: Animal in box at 1:45 p. m., temperature 102; out at 3 p. m., temperature 101.

January 28: In box at 8:50 a. m., temperature 102.4; out at 9:50 a. m., temperature 101.6.

January 28: In box at 2 p. m., temperature 102; out at 3:15 p. m., temperature 101.5.

January 29: In box one and one-half hours, temperature before, 102.4; after, 100.

January 30: In box one and one-half hours, a. m.; temperature before, 103; after, 102. January 30: In box one hour p. m.; temperature before, 103.2; after, 100.4; diarrhea and vomiting followed.

January 31: Dog not so lively. Three hours after last inhalation found lying down most of time; shivering on attempt to stand; weakness in hind legs is manifest. Weight 6 kg. (13 pounds). Pupils widely dilated, 8 mm., and not reacting to medium light. After prolonged ophthalmoscopic examination they react to from 4 to 5 mm. Fundi: arteries thin, veins black, disks pale.

February 2: In box at 10:50 a. m., temperature 102; out at 1:30 p. m., temperature 98; unconscious when taken out of box and was semicomatose for about three hours. Some reddish diarrheic stools containing blood. Pupils dilated to 10 mm., only the rim of iris showing. No reaction to light. At 4:30 p. m. pilocarpin 2 per cent. solution instilled into eyes. At 4:50 p. m. no response to miotic. Fundus O. D., optic disk pale; O. S., temporal pallor of optic disk.

February 4: In box at 8:45 a. m. with 5 ounces methyl alcohol. Temperature 102 F.; taken out of box at 11:15 a. m., temperature 97.4; animal unable to stand.

February 5: Weakness in legs on standing.

February 7: Weight of animal 5 kg., 840 gm.; O. D. disk slightly pale, O. S. disk pale but not so pale as in O. D.

February 9: In box at 1:25 p. m., temperature 100.2; out at 2:45 p. m., temperature 100.

February 10: In box at 9:45 a. m., temperature 101.6; out at 11 a. m., temperature 100.2. Gait unsteady; eyes red; frothing at mouth.

February 11: In box at 1:25 p. m., temperature 101.2; out at 2:25 p. m., temperature 99; weak and sluggish; frothing at mouth.

February 12: In box at 8:35 a. m., temperature 101.2; out at 9:35 a. m., temperature 99. In box at 2:50 p. m., temperature 101.8; out at 4:05 p. m., temperature 98.4

February 13: In box at 9:45 a. m., temperature 102; out at 10:45 a. m., temperature 100; sluggish with mucus in throat. At 3 p. m. in box, temperature 100; out at 4 p. m., temperature 99.6.

February 14: At 4 p. m., in box, temperature 101; at 4:45 p. m., out, temperature 100.4; optic disks same as when animal was last examined.

February 16: At 12:25 p. m., in box, temperature 102.6; out at 1:25 p. m., temperature 100; sluggish.

February 17: At 2:55 p. m., in box, temperature 102; out at 3:55 p. m., temperature 100; not much affected. Weight 6 kg., 880 gm., after eating.

February 18: At 2:25 p. m., in box; temperature 102; out at 3:25 p. m., temperature 100.4; shows staggering gait.

February 19: At 9:40 a. m., in box; temperature 101.2; out at 10:40 a. m., temperature 98.6; condition of animal good but gait is staggering.

February 20: At 9:20 a. m., in box; temperature 101; out at 10:25 a. m., temperature 99; staggering gait.

February 21: At 9:40 a. m., in box; temperature 100; out at 10:40 a. m., temperature 98; condition good, but trembles and has staggering gait. At 4:40 p. m., temperature 101.4.

February 24: At 9:25 a. m., in box; temperature 101; out at 10:25 a. m., temperature 99. At 4 p. m., ophthalmoscopic examination: O. D., pallor of disk; right eye enucleated under chloroform anesthesia for microscopic examination (chloroform given in error by assistant instead of ether);

4:10 p. m., dog died during examination, very shortly after few inhalations of chloroform vapor; only exceedingly small quantity used.

Necropsy: Spleen rather dark blue, not as deep color as found in acute cases; stomach, patches of congestion; other organs apparently normal.

*Remarks.*—The intention to administer to the animal wood alcohol by inhalation over a long period was only partially carried out. The animal died after a few whiffs of chloroform (given by mistake instead of ether), a fact which showed its lowered resistance. Although frequently affected by the inhalation of the wood alcohol, it recovered after each inhalation during the early stages of the experiment. Later the effect of the inhalations began to be manifested in the appearance of vomiting, bloody diarrhea, general weakness and loss of appetite. The dilatation of the pupils, their loss of reaction to light and to miotics, the edema and later the pallor of the optic disks and the congestive patches of the gastric mucosa are some of the interesting features shown in this experiment.

EXPERIMENT 10.—Changes in the electroconductivity in the blood of a dog produced by methyl alcohol inhalation.

Jan. 17, 1914: Dog, weight 5 kg. 570 gm. (11¾ pounds). Under ether anesthetization 15 c.c. of blood were obtained to which was added 5 c.c. of a 1 per cent. aqueous solution of ammonium oxalate. The electroconductivity was measured by Edelmann's Universal Widerstands Brücke: see technic.[1]

(1) Sample of dog's blood mixed with ammonia oxalate 3 to 1; (2) bridge reading, 39.4; (3) resistance out, 100; (4) temperature 9° C. (48.2 F.) = 65.017 ohms resistance at 9° C.; = 54.615 ohms resistance at 17° C. (62.6 F.).

January 20: Dog kept in box three hours with fumes from 5 ounces wood alcohol. Respirations 42, labored; at end of time lies on right side unconscious. Temperature below 94 F. Eyes: cornea slightly hazy; corneal and pupillary reflex abolished; fundus, edema of disks with dark veins; arteries small.

Blood was obtained from which the bridge reading was 34; resistance out 100; temperature 17 C. = 51.515 ohms resistance at 17 C. Dog killed with chloroform; eyes and optic nerves were enucleated and preserved in formaldehyd solution.

Post-mortem Examination.—Pericardium contained large amount of colorless transparent fluid which spurted out as if under pressure when it was incised; macroscopically heart appeared normal; stomach and intestines were dilated; interior of stomach and pylorus very markedly congested; cupful of frothy mucus found in stomach; spleen very dark indigo blue.

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[1] Centralbl. f. Electric., 1887, p. 115.

*Remarks.*—This experiment showed that the electroconductivity of the serum was increased after the inhalation of wood alcohol. The difference of 3.1 ohms resistance obtained in testing the electroconductivity of the blood showed a change which might result from the breaking up of the corpuscles, from an increase in H-ion content, or from any increase in the inorganic salts. An increase in the H-ion content will produce an increase in the acidity of the blood and also conduces to an edema of the tissues. As we find evidence in the blood of acidosis, and a tendency to edema of the ocular tissues, we think that the increase of its electroconductivity, following methyl alcohol poisoning, is due to an increase in the H-ion content of the blood.

EXPERIMENT 11.—Acute poisoning by inhalation in a monkey. Jan. 22, 1914: Female monkey in good health, weight 4 kg. (8.8 pounds), 1 per cent. solution of atropin instilled into eyes. Apparently did not like the bitter taste, as it evidently passed through nasal duct into mouth. It kept expectorating and washing out its mouth; gagged several times as if nauseated. Eyes: pupils dilated quickly and at maximum; fundi, periphery very dark, uniformly pigmented, no abnormalities; disks appear normal and are oval, long diameter vertical.

January 29: Temperature 102 F. per rectum. Monkey placed in box with 3 ounces methyl alcohol spread over sides of box. At 4:25 p. m. air pumped in under 2½ pounds pressure thirty times per minute. Monkey sat in one corner opposite air inlet and no apparent intoxicating effect was produced whatever; at 5:10 p. m., wood alcohol, 1 ounce additional introduced into box; at 5:25 p. m., taken out; monkey acts normally.

February 3: General condition good. At 5 p. m., in box with 3 ounces wood alcohol; same condition; temperature 100.6; at 5:50 p. m., taken out, temperature 100.2 F.

February 4: At 1:35 p. m., in box; temperature 101.4; out at 3:15 p. m., temperature 102; staggering gait, acted dazed and slightly intoxicated. Three ounces wood alcohol used, box with open ends.

February 5: At 2:55 p. m., in box; temperature 102; out at 4 p. m., temperature 100.6. Three ounces wood alcohol used. At 3:35 p. m., animal winking; slight watery discharge from nose.

February 6: At 2:45 p. m., in box; temperature 101.6; out at 3:45 p. m., temperature 101.6.

February 7: At 3:05 p. m., in box; temperature 102; out at 4:30 p. m., temperature 102; 3 ounces wood alcohol placed on cotton, high up, near top of box; monkey not affected apparently by vapor.

February 9: At 3:15 p. m., in box; temperature 102; out at 4:15 p. m., temperature 102; no effect. Wood alcohol 3 ounces.

February 10: In box at 2:55 p. m., temperature 102; out at 4:40 p. m., temperature 100; pulse 160; optic disks hyperemic as compared with first examination; veins dark; pupils dilated, sluggish to light, acts sleepy. Wood alcohol 3 ounces.

February 11: At 3 p. m., in box; wood alcohol 3 ounces; temperature 102; out at 4 p. m., temperature 101.6; staggering gait, weak hind legs; eyes red and watery; drowsy, falls forward in attempting to walk.

February 12: At 10:20 a. m., in box; temperature 101; out at 11 a. m., temperature 99. Wood alcohol 3 ounces.

February 12: At 4:10 p. m., in box; temperature 103; out at 4:20 p. m., temperature 102; vomited, much affected, scarcely able to walk; hygrometer 65.

February 13: At 9:45 a. m., in box with 5 ounces wood alcohol; temperature 100.4; out at 10:45 a. m., temperature 100; drowsy; much affected; very weak, nausea, vomiting. At 4 p. m., temperature 98.8; acts ill, face, gray pallor, refused to eat chocolate, of which was very fond. Keeps quiet; eyes red, pupils dilated, surface of body cold; wrapped up in covers, sits with head down between fore legs; placed in room by warm radiator.

February 14: At 8 a. m. found dead; body warm, apparently just died. No evidence of painful death. Head removed, eyes enucleated. Necropsy at 5 p. m. macroscopically. Pancreas and adrenals normal; kidneys slightly congested; liver slightly darker than normal, uniform color, no apparent degeneration; uterus and ovaries normal. Heart, pericardium slightly congested; empty; no blood clots nor fluid. Lungs normal, crepitate and float; spleen small, indigo color characteristic of wood alcohol cases; preserved in formaldehyd solution. Large intestines very much inflated containing also mass of feces and a thrombus in the intestinal mucosa. Small intestines not much bloated; empty of fecal matter but in upper part sections or links bloated with occasional dilatation of vessels; stomach contains yellowish-green tenacious semifluid homogenous mass of mucus with small pin-point hemorrhages along vessels, with dilated veins; dura markedly and pia mater slightly congested; spinal cord appeared normal.

*Remarks.*—The shortness of time of inhalation with free ventilation, and the small amount of wood alcohol used, explain the lack of effect on the monkey during the first two exposures. But this lack of effect was only apparent, as the animal began to show weakness of hind legs, loss of appetite, diarrhea and vomiting after the subsequent exposures. That death followed the last exposure, in which 5 instead of 3 ounces of wood alcohol were used, is very significant. We are convinced that a latent debilitated condition which was not recognized, existed, and the last dose acted fatally as an overdose. The pin-point hemorrhages on the gastric mucosa is one of the characteristics of wood alcohol poisoning.

EXPERIMENT 12.—Electroconductivity of the blood of a dog after inhalation of methyl alcohol.

Feb. 24, 1914: Dog, male, weight 6 kg., 500 gm. At 10:35 a. m., in box with 5 ounces methyl alcohol; temperature 103; out at 2:35 p. m., temperature 95; respirations 32; pulse 130. Ophthalmoscopic examination: Hyperemia and edema of disks with dark vessels; left eye enucleated for microscopic examination. Dog killed by chloroform. Spleen very dark indigo-blue with mulberry excrescences on it (resembling those found in typhus fever). Electroconductivity of blood: bridge reading 23½; resistance out (3 posts), 100; temperature 10 C. (40 F.) = 30.719 ohms resistance at 10 C.

*Remarks.*—The blood of this animal after exposure to the vapor of methyl alcohol showed a very low ohms resistance, consequently a very high electroconductivity.

EXPERIMENT 13.—Acute poisoning in dog by inhalation of Columbian spirits, with marked ocular hypotension. Feb. 28, 1914: Dog, weight 5 kg., 900 gm., male. At 8:35 a. m., in box with 10 ounces Columbian Spirits; temperature 102.6; out at 11:15 a. m. In box at 12 m., out at 3:20 p. m, temperature 96. In box six hours; during first hour frothed at mouth; required about one hour to be intoxicated, then comatose; at 4 p. m., respirations 28; auscultation showed short inspiration with long expiration; pulse 88 by stethoscope; temperature below 93. Some fibrillary contractions of muscles of face, mouth, neck, nose and tongue. Intraocular pressure less than 3.5 mm. Hg with Schiötz tonometer. Not much bleeding during enucleation, but more than when commercial wood alcohol was used; bleeding stopped readily. (viscosity increased, coagulation time reduced). Died one hour later as expected, using the marked hypotension as an indication of the proximity of death. The examination of the ocular fluids showed a marked reduction in alkalinity.

Experiment 14.—Changes in chemical reaction of serum and aqueous humor. March 3, 1914: Female dog, weight 5 kg., 800 gm. Aqueous humor withdrawn by aspiration under cocain anesthesia. Four-tenths c.c. obtained from left eye. Alkaline: 48 — 47.90 = 0.10; acid: 28.10 — 28 = 0.10; result, neutral to phenolphthalein.

March 5, 1914, 10 a. m.: In box; temperature 101; out at 12:30 p. m. In box 12:45 p. m., with 5 ounces Columbian Spirits; out at 4 p. m., temperature 95. Eyes: pupils 3 mm.; 5 p. m.—O. D., tonometer = 3.5; O. S. = 3.5; (blood examination morning and evening). Died next day as indicated by very low hypotension.[2] Right eye, equeous 0.4 c.c. Titrated with phenolphthalein as an indicator. Alkaline: 32.4 — 29.5 = 2.90; Acid: 31.10 — 29.95 = 1.15; · Alkaline: 2.90 — 1.15 acid = 1.75 alkaline = 4 c.c.; 4.48 tenth normal sulphuric acid to 1 c.c. Normal blood-serum 0.1 acid = 1 c.c. Blood normal 0.5 c.c. Twenty c.c. water. Alkaline: 33 — 32 = 1.00; acid: 31.95 — 31. = .95; alkaline 1.00 — acid 0.95 = .05 alkaline. One c.c. = 0.1 blood tenth normal sulphuric acid to phenolphthalein. Serum taken in morning.

Blood after exposure to Columbian Spirits vapor: Alkaline: 23 — 22 = 1.00; acid 32.85 — 32.00 = .85; alkaline 1.00 — acid .85 = .15 alkaline. One c.c. blood-serum[3] = 0.15 tenth normal sulphuric acid.

*Remarks.*—As we see, the serum of the dog before inhalation was 0.10 tenth normal sulphuric acid; aqueous humor was neutral; serum after inhalation was 0.15 tenth normal sulphuric acid; aqueous after inhalation was 4.48 tenth normal sulphuric acid. It is very significant that the aqueous shows more acidity than the blood.

Experiment 15.—Control. Feb. 3, 1914: Male dog, weight 3 kg., 300 gm., was placed in box as control with inlet and outlet open (no methyl alcohol vapor added). Animal was kept in box four hours. No apparent effect; was active and lively when taken out.

February 7, 9:55 a. m.: In box, temperature 103.4; out at 1:15 p. m., temperature 102.6; box closed tight, no ventilation; when taken out box contained bad (close) odor. Dog showed no bad effect.

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[2] Tension when less than 5 usually dies within twenty-four hours.

[3] Blood-serum 1 c.c. = 0.05 increased acid in case after Columbian Spirits.

EXPERIMENT 16.—Acute poisoning with Columbian Spirits showing acidity of aqueous humor and increased viscosity of blood. March 7, 1914: Male dog, weight 6 kg. (about 12 pounds), in box with 10 ounces Columbian Spirits. At 9:30 a. m., in box; temperature of dog 104; out at 1:45 p. m., temperature 97. At 2:45 p. m., in box; temperature 95; out at 4:15 p. m., temperature 94. Pupil contracted to 2 mm.; no reaction to light; dog comatose; aqueous withdrawn with needle, 0.7 c.c. Alkaline: 24.00 — 23.00 = 1; acid 34.85 — 34.00 = 0.85; alkaline, 1.00 — acid .85 = .15; 0.7 c.c. = 0.15. One c.c. = 0.21 tenth normal sulphuric acid to phenolphthalein as indicator.

*Remarks.*—As normal aqueous showed neutral reaction, this test is interesting on account of acid reaction as determined.

March 9, 9:30 a. m.: Temperature 96.4; animal comatose; pulse 20; respiration 6. Pupils 2 mm., contracted; no light reaction. Corneal reflex abolished, anesthetic; O. D., fundus hyperemic and edema of optic disk; arteries small, veins not altered in size. At 4:30 p. m., temperature 95 F.; respiration 6; pulse 20. O. D. enucleated and placed in 5 per cent. formaldehyd solution for microscopic examination, no bleeding during operation until optic nerve was severed, then slight hemorrhage occurred, about 1 dram dark viscous blood which coagulated readily. Surface of body cold; respirations slow and deep; eyes closed; tongue dry and protruding; animal comatose; pupil, no light reaction—tonometer 3 by palpation, all reflexes abolished; animal in moribund condition.

March 10: Found dead at 8 a. m. Necropsy: Brain reddish; meninges and substance of brain (white and gray matter) congested. Lungs, pleural cavity contains about 2 c.c. sanguineous fluid, surface of left lung shows numerous small hemorrhages; lung substance slightly edematous and floats on the surface of water. Trachea, normal, not congested. Heart, full of dark blood-clots. Spleen, dark indigo-blue throughout. Liver and bile both darker than normal. Kidneys, bluish-red, very dark, much congested. Adrenals and pancreas darker than normal. Stomach, empty; numerous small punctate dark purple hemorrhages along lesser curvature. Intestines, bloated and empty, purplish-red, similar to lining of stomach.

*Remarks.*—The symptoms, chemical reactions of the aqueous and post-mortem findings we consider typical of acute poisoning with methyl alcohol.

TABLE OF RESULTS OF EXPERIMENTS ON ANIMALS FOLLOWING THE ADMINISTRATION OF METHYL ALCOHOL

| | Kind of Animal | Length of Experiment, Days | No. and Kind of Exposures | Amount and Kind of Wood Alcohol Used | Main Symptoms | Ocular Symptoms | Remarks |
|---|---|---|---|---|---|---|---|
| 1 | Dog | 30 | 4, no ventilation | Wood alcchol, 16 oz. | Continuous movements of head from side to side, uncertain gait, drop of temperature, twitching of extremities, coma. | Congestion and edema of disks. Dark blood-vessels. Pupils dilated, not reacting to light. | Death after 4 exposures. |
| 2 | Guinea-pig | 5 | 2, no ventilation | Wood alcohol 6 oz. | Convulsive resp.; uncertain gait. Unconscious after second exposure. | .................... | Death after second exposure. |
| 3 | Two guinea-pigs | 1 | 1, no ventilation | Wcod alcchol 10 oz. | Intoxicated; semicomatose | .................... | Mucosa of stomach shows a red, rough area. |
| 4 | Guinea-pig | 5 | 2, no ventilation | Columbian Spirits, 4 oz. | Occasional convulsive movements of body and lateral motion of head; weakness of hind legs. | .................... | Death after two exposures. Dark suprarenal glands, with hemorrhage in one. Multiple hemorrhages in the stomach mucosa. |
| 5 | Dog | 13 | 5, 4 with ventilation, 1 with no ventilation | Col. Spirits, 11 oz. alcohol, 18½ oz. | No apparent effect when ventilation free. Drop of temperature. Coma, when no ventilation. | Very low intraocular pressure during coma. Congestion and edema of disks. | Death only after exposure without ventilation. |
| 6 | Rabbit | 15 | 4, no ventilation | Columbian Spirits, 18 oz. | Spasmodic contraction of body and diaphragm. Drop of temperature and coma after fourth exposure. | Loss of pupillary reaction and of lid reflex in coma. | Seems to be less susceptible. Died only after the 4th exposure to relatively large dose. |
| 7 | Guinea-pig | 1 | 1, subcutaneously | Columbian Spirits, 5 c.c. | Instantaneous coma; convulsions. | Persistence of corneal reflex. | Death in 68 min. Persistence of heart action long after respiration ceased. |
| 8 | Dog | 1 | 1, no ventilation | 5 oz. | Coma, labored respiration. Prolonged expiration, staggering gait, frothing at mouth. | Corneal sensibility diminished, no pupillary reaction, pupils slightly contracted, disks congested, edema, veins, dark; arteries, thin. | Death about 36 hours after one prolonged exposure. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 9 | Dog | 48 | About 40, no ventilation. | 200 oz. | Drop of temperature; shivering; diarrhea; vomiting. Animal comatose after 3 weeks' exposure, recovers, staggering gait. | Pupils dilated, no reaction. Disks, pink; arteries, small; veins, dark; diminished corneal reflex. Later pale optic disks, dark veins. | Exposures lasted only 1 to 2 hours at a time. Died from a few whiffs of chloroform. Postmortem: Patches of congestion in mucosa of stomach. |
| 10 | Dog | 3 Hours | 1, no ventilation | Wood alcohol, 5 oz. | Labored resp., coma. | Abolished corneal and pupillary reflex. Edema and congestion of disks; arteries small, veins dark. | Electroconductivity increased. Post mortem: Large amount of serum in pericard. Patches of congestion in stomach, black-blue spleen. |
| 11 | Monkey | 22 | 12 with ventilation | 38 oz. | Drop of temperature; slight symptoms of intoxication after 3 exposures; weakness of hind legs; vomiting. | Hyperemia of optic disks after each exposure. Sluggish reaction of pupils to light. | Died from overdose. Five instead of 3 ounces wood alcohol. Post mortem: no blood in heart, spleen dark blue. Pin-point hemorrhages in stomach. |
| 12 | Dog | 1 | 1, no ventilation | Wood alcohol, 5 oz. | Drop of temperature. Coma. | Hyperemia of optic disks. Dark blue vessels. | Electroconductivity of blood increased. Post mortem: Dark blue spleen as in typhus. |
| 13 | Dog | 1 | 1, no ventilation | Columbian Spirits, 10 oz. | Coma after one hour exposure. Prolonged expiration. Drop of temperature. Fibrillary contractions in face, neck, legs, chest. | Very marked drop of intra-ocular pressure. | Increase in acidity of the blood and intra-ocular fluids. Rapid coagulation of blood, viscous appearance. Very little bleeding during enucleation. Rapid death after the ocular hypotension set in. |
| 14 | Dog | 1 | 1, no ventilation | Columbian Spirits, 5 oz. | Drop of temperature. Coma. | Marked ocular hypotension. | Reduced alkalinity by increased acidity, much more in the ocular fluids than in the serum. |
| 15 | Dog | 4 | 2, closed box, no wood alcohol | None | .................... | .................... | .................... |
| 16 | Dog | 3 | 2, no ventilation | Columbian Spirits, 10 oz. | Coma. Very low rate of pulse and respiration. Drop of temperature. | Pupils contracted; no light reaction; hypotension marked; hyperemia and edema of optic disks. | Viscosity of blood; increased acidity of aqueous. Post mortem: congestion of brain, multiple hemorrhages surfaces of lung, spleen indigo blue; punctate hemorrhages of the stomach mucosa. |

## BLOOD-COUNT IN DOG WITH ACUTE POISONING BY INHALATION OF METHYL ALCOHOL

Before inhalation:

| | |
|---|---|
| Erythrocytes | 5,700,000 |
| Leukocytes | 11,800 |
| Hemoglobin | 90 per cent. |
| Polymorphonuclears | 79 per cent. |
| Lymphocytes | 21 per cent. |

After inhalation (2½ hours):

| | |
|---|---|
| Erythrocytes | 7,800,000 |
| Leukocytes | 28,600 |
| Hemoglobin | 100 per cent. |
| Polymorphonuclears | 99 per cent. |
| Lymphocytes | 1 per cent. |

*Remarks.*—Besides the increase in the erythrocytes, leukocytes, hemoglobin and polymorphonuclears, the very marked decrease in the lymphocytes is of decided interest.

## MICROSCOPIC FINDINGS IN EYES OF DOG WITH SUBACUTE POISONING

### HEMATOXYLIN EOSIN AND VAN GIESON

1. Choroid gives the impression of hyperemia.

2. Tapetum normal. Retina: rods and cones and the external nuclear layer normal.

3. Outer reticular layer slightly edematous.

4. Inner nuclear layer, normal.

5. Inner reticular layer, slight edema.

6. Ganglion cells swollen and surrounded by clear areas, the nuclei being displaced far toward the periphery.

7. Inner layers of the retina show some edema.

### WEIGERT

1. The medullary sheath of the optic nerve takes Weigert's stain rather weakly.

2. A few vacuoles show at the ends of the rods and cones.

The Marchi and Nissl specimens have not been finished, the report of these will be published later.

We consider methyl alcohol to be a true hematotoxic. It has a decided effect on all tissues, producing degenerations, and under certain conditions amblyopia or death. In the eye it produces first a toxic edema of the tissues, which with the increased viscosity of the blood, interferes with its free circulation. The tissues, being deprived of their nutrition and oxygen, succumb to the effect of the toxin, and degeneration of the ganglion cells and nerve fibers ensues. Acidosis occurs as indicated by the results of the titration of the aqueous and serum, and by the determination of the electroconductivity of the blood.

The tension of the eye in methyl alcohol coma may be a guide as to the immediate outcome of the case, and with more study a scale might be formulated giving exact points at which, as regards life, one could prognosticate complete recovery or death.

Hypotension of 5 mm. Hg or less indicates death within twenty-four hours, while with tension over 5 the animal lived longer; and in these cases a question arises whether or not, if proper treatment was instituted early, the animal would recover.

## SUMMARY

Effects of experiments with methyl alcohol inhalation were the following:

1. In nose and mouth copious discharge and frothing.
2. Temperature: marked fall, varying from 1 to 10 degrees according to amount used, method and time of exposure to vapor.
3. Respiration: rate increased, then decreased below normal, being very low just prior to death and ceasing before heart.
4. Blood: increase in electroconductivity due to an increase in H-ion content. Increased acidity, with phenolphthalein as an indicator. Coagulation time reduced, specific gravity increased. Viscosity increased. Erythrocytes, leukocytes, hemoglobin and polymorphonuclears increased and lymphocytes decreased.
5. Necropsy findings: Stomach: empty, mucous membrane congested, punctate hemorrhages. Trachea: pale. Lungs: few minute hemorrhages on surface. Heart: muscle dark, in some cases cavities, empty. Liver: dark. Kidneys: dark purple, congested. Adrenals: hemorrhage in one case. Spleen: dark indigo-blue. Intestines: bloated, small punctate hemorrhages. Brain: marked congestion of the meninges.

Effects on the eyes were: Slight irritation producing blinking, slight haziness of cornea and no inflammation of conjunctiva. Corneal reflex retained in some animals after others had disappeared. Fundus: Hyperemia and edema of the disks, with dilated, dark veins and arteries. Pupil: Moderate contraction in coma, with loss of light reaction. Tension varies with intensity of toxemia, in mild recoverable cases being normal, and in profound coma showing marked hypotension, which when less than 5 mm. Hg by tonometer, indicated a rapid and fatal termination within about twenty-four hours.

Aqueous by titration (with phenolphthalein as an indicator) showed an acid reaction.

In one dog (Experiment 9) blindness was noticed, judging from the animal's collision with objects when attempting to walk, as he could not see to get around them.

Microscopic findings indicate an edema of the tissues with very early signs of beginning degeneration of the ganglion-cell layer of the retina.

In reviewing the results of these experiments, attention is directed to the similarity with those obtained through experimental researches and clinical observations, in the internal administration of methyl alcohol; thus demonstrating its fatal and profound toxicity irrespective of the method of its entrance into the animal organism.

The contentions of the manufacturers that the refined methyl spirits are absolutely harmless are untenable and may cause many sad results among workmen by rendering them careless in taking proper precautionary measures while working with it. Many consider its inhalation harmless under ordinary ventilation. We take exception to that supposition. Most of our inhalation clinical cases result from its toxicity under what are considered by the workmen ordinary conditions. While ventilation will diminish the intensity of the toxicity, the inhalation will not be rendered absolutely harmless thereby. Not ordinary ventilation, but the very best obtainable is required for workers using wood alcohol. At least three times the ordinary air-space allotted per person should be allowed to each workman using it. Placards warning of the poisonous properties of wood alcohol should be posted in conspicuous places about premises where it is used, and hours of labor should be so arranged as to allow for exercise in the open air at frequent intervals. In our subacute cases while the exposure to the vapor for an hour or two daily had not much apparent untoward effect at the time, it certainly did have an effect which manifested itself later by subsequent weakness, trembling and unsteady gait in the intervals between exposures. We are inclined to agree with M. L. Poincaré,[4] who found, after exposing animals to small quantities of vapor of methyl alcohol over a period of from eight to sixteen months, that considerable fatty degeneration of tissues occurred, especially in the liver, heart, muscle, epithelial cells of the uriniferous tubules, some cells of the lungs, and with congestion and slight inflammation of the meninges, and of some nerve centers. He concluded that the vapor of methyl alcohol in small quantities was dangerous.

Physicians often fail to recognize industrial and occupational diseases because of the fact that they are often masked under the form of chronic disorders, which show themselves in obscure diseases of the digestive tract and nervous system, and are attributed by patients to other causes. A number of cases of methyl alcohol poisoning, to our knowledge, have occurred in which the causes of death were given as pneumonia, meningitis, heart-disease, etc. Many of the milder cases have been treated for chronic gastritis, hepatitis, etc. It is not surprising that these unfortunate persons travel from one physician to another searching for relief, when we consider the unfortunate but sad truth that many physicians fail to grasp the true etiology of the cases.

That a difference in susceptibility to methyl alcohol exists in animals and man cannot be denied, and this can be partly explained by the rate of oxidation which takes place in the animal or person. Those in which it is rapidly oxidized experience the least effect, while those with slow oxidation feel the greater toxic effect. Harnack[5] concluded that methyl alcohol in slow oxidation formed formic acid, while in rapid oxidation carbon dioxid and water were formed. Hence when it is rapidly oxidized, it is comparatively (?) harmless, while in slow oxidation, which usually occurs in man, it is exceedingly toxic, as the action of the formic acid thus produced in the animal organism was much stronger than that of the pure formic acid introduced into the body, as in the latter case the organism attempts to convert it with bases and thus gives rise to formates.

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[4] Poincaré: Compt. rend. Acad. d. sc., 1878, p. 682.

[5] Harnack: Deutsch. med. Wcbnschr., xxxviii, 358.

In 1869 Richardson[6] suggested that methyl alcohol be inhaled as an anesthetic. He stated that 2 ounces required one hour to produce anesthesia, and pointed out that reflex irritability was not abolished by small doses, that the temperature of the body was reduced 3 degrees and that the danger from its use was practically nil; but when inhalation was forced, death was produced by simultaneous cessation of respiration and circulation. We do not know the time required for him to gain the knowledge contained in the last sentence, but trust that it was during his very early use of methyl alcohol.

It is a well-recognized fact in pharmacology that poisons which affect the highly differentiated nerve-structures very powerfully are proportionately more dangerous the more highly developed the nervous system of the animal is, hence it would be expected that man would be relatively more susceptible to such a poison as methyl alcohol than would the dog or rabbit.

The results of the experiments with the titration method agree with the conclusions formed by Gregnolo,[7] who determined, after the ingestion of methyl alcohol, that there was an increase in the concentration of H-ions in the aqueous and serum, and who stated that these changes go parallel with the morphologic changes in the tissues of the eye. His experiments show, as do ours, that the substances produced in the body by poisoning with methyl alcohol are extremely diffusible and penetrate the eye readily. Our microscopic specimens having been obtained from acute cases show only the stage of edema with an indication in the ganglion cells of one of the first signs of degeneration, the location of the nucleus being far toward the periphery of the cell and most of the cells showing this change. As to the fate of methyl alcohol in the human body, Pohl[8] found that it underwent the same change as in that of the lower animals, its administration leading to the appearance of formic acid which is slowly excreted in the urine.

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[6] Richardson: Med. Times and Gaz., 1869, p. 703; London Med. Rec., 1875, pp. 62-77-93.

[7] Gregnolo: Klin. Monatsbl. f. Augenh., 1913, p. 157.

[8] Pohl: Arch. f. exper. Path. u. Pharmakol., 1893, p. 181.

R. Hunt[9] states that experiments on the physiologic action of methyl alcohol show conclusively that, however pure the preparation may be, it is totally unfit for use as a substitute for grain alcohol in any preparation which is to be taken internally or used externally, or, we add, which may be inhaled in a concentrated form.

### USE OF SPECIALLY DENATURED ALCOHOL RECOMMENDED

Only those who have had experience realize the power and intensity of the opposition encountered in endeavoring to obtain proper protective legislation for the regulation of the sale and use of methyl alcohol. When it is possible for one or two members of a legislative body working in the interests of methyl alcohol manufacturers to be able to thwart the wishes of the vast majority of the inhabitants of a great state for a just protective law regulating the safe and proper labeling of methyl alcohol, it is a most unjust and deplorable state of affairs. If the law regulating the sale of specially denatured alcohol could be amended so that manufacturers might be permitted to sell this alcohol to the individual trades using it in the various industries, which would allow them to prepare their own varnishes, etc., obtaining the consistency desired, one of the objections to its present use would be overcome. The variety of denatured alcohol which the law now compels them to use contains a denaturing agent which renders the varnish of soft consistency, and after having dried it scratches very easily, leaving a white mark. If the trades could obtain the denatured alcohol without the objectionable denaturing agent (benzin, pyradin, etc.) it would in a short time supplant the methyl alcohol to a great extent. As it is only one-tenth as dangerous and if used in an exceedingly well-ventilated space, the fatal results due to the toxicity of methyl alcohol would be reduced to a minimum.

The laws relating to methyl alcohol should be enforced with a jail sentence as a penalty attached. Until the gravity of the situation is realized by our law-makers and the judges in our courts, a certain number of manufacturers who value human life cheaply will continue to be a menace to society by disregarding the law and thus causing blindness and death.

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[9] Reid, Hunt: Bull. Johns Hopkins Hosp., xiii, Nos. 137-138.

### TREATMENT

While heretofore treatment, except in a very small proportion of the cases of methyl alcohol poisoning with amblyopia, has been considered rather hopeless, the few patients who have improved or recovered, should stimulate us in our efforts to go forward with renewed vigor in search of a plan of medication which shall yield better results. To formulate a plan of treatment based on our findings might be summed up in two words, eliminate and stimulate. Eliminators of all varieties should be used, namely, emetics, hydragogue cathartics, diuretics and diaphoretics, calomel, jalap, pilocarpin hypodermatically, hot saline baths or packs, saline infusions and high saline enemas. Stimulants such as strychnin, caffein and theobromin would produce stimulation with diuresis. Ammonia and oxygen inhalation may be used to supply the alkalines and to aid in oxidation. Sodium iodid and alkaline waters would aid in reducing viscosity of the blood, and the medicinal treatment may be supplemented by applying heat to the body and extremities.

Credit is hereby given and our sincere thanks extended to Professors Lee, Opitz, Scott and Pike for their kindly assistance and advice tendered in these researches, which were made in the physiological laboratory of the College of Physicians and Surgeons, Columbia University, New York, also to Mr. Edward Burchell, for his kind assistance in preparing and mounting the microscopic specimens and to Drs. Dixon and Holden for their kindness in examining our microscopic specimens.

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# MEMORANDA

# MEMORANDA

# ENUCLEATION WITH TRANSPLANTATION OF FAT INTO THE ORBIT*

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The loss of an eye is to any one a dire calamity, but to the laborer or artisan it has its economic side as well, since employers are usually adverse to giving employment to a man who possesses but one eye.

It becomes our duty therefore when called on to remove an eye to provide for a cosmetic after-result that will simulate as nearly as possible a normal appearance, and to strive not only for mobility of the artificial eye but to provide as well a sufficient cushion or stump for it to rest on and thus do away with the tell-tale depression of the upper lid, so conspicuous in simple enucleations.

For the past two years and a half I have transplanted fat into the orbit in all of my cases of enucleation, sixty-six in all.

In the last twenty-six cases I have departed from the usual technic as originally proposed by Barraquer, since with it I noticed too frequently a shrinkage of the cushion from absorption of the fat. I reasoned that the old technic which required the fat to be enclosed within Tenon's capsule provided for entirely too little fat, and also that the fat was not favorably placed to secure the best nourishment. The success with the improved technic, which calls for a much larger piece of fat and direct union of the fat to the conjunctiva, has been most gratifying.

I divide the conjunctiva close to the limbus, dissecting it and Tenon's capsule back as far as possible. A strabismus hook is then introduced under the superior rectus, which is held by an assistant while a double-armed No. 00 twenty-day chromic catgut suture is

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* This paper has been accepted by the Executive Committee of the Section on Ophthalmology of the American Medical Association, to be presented before the Section at the Atlantic City Session, June, 1914. Publication rights reserved by the American Medical Association.

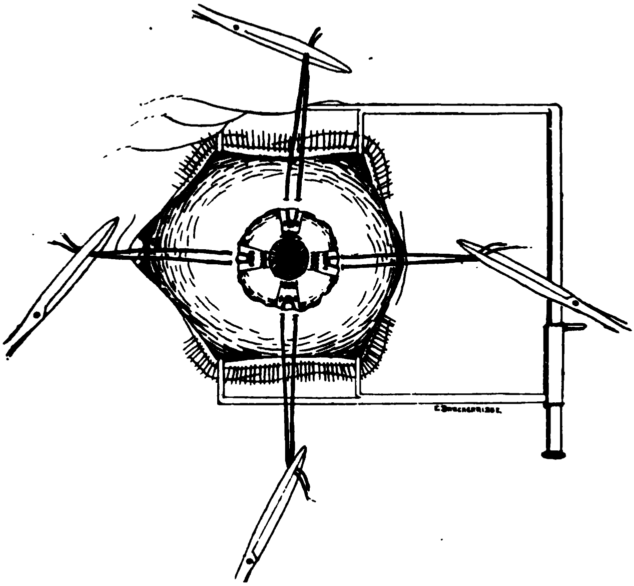


Figure 1.

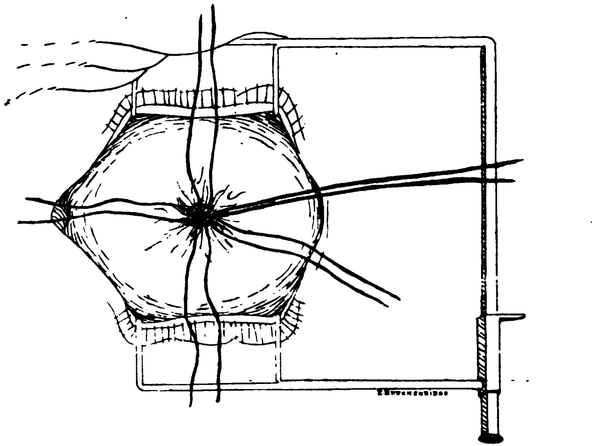
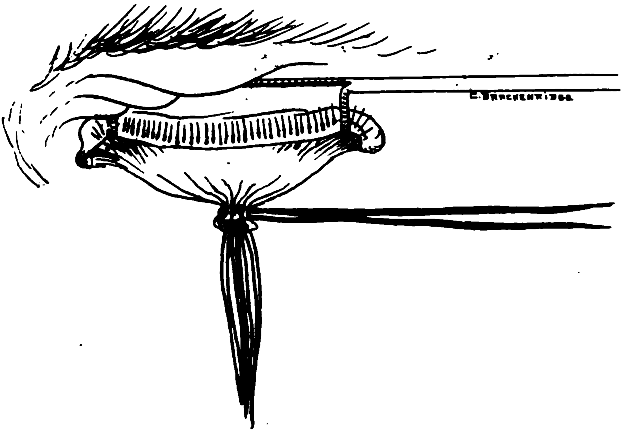


Figure 2.

passed through the muscle from below upward close to its lateral margins behind the hook and loosely tied on top. The suture is then passed through the conjunctiva about 2 mm. from its edge directly over the muscle; it is not tied at this time, but only disarmed and the ends held in a pair of hemostats. The internal, inferior and external recti muscles with their overlying conjunctiva are similarly treated (Fig. 1). The eyeball is then forced out of the socket by pressing the speculum backward, drawn toward the nose, and the optic nerve severed. The oblique muscles and any adherent tissues are severed and the socket turned



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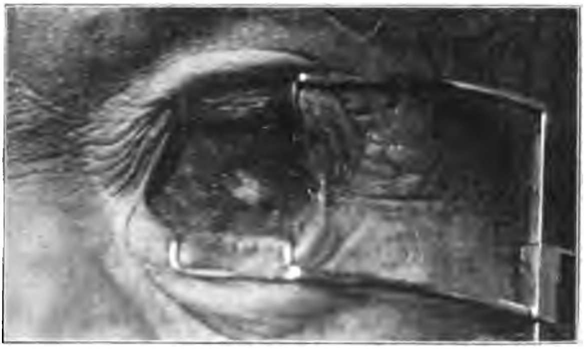

Figure 4.

over to an assistant, who, making gentle traction on the four catgut sutures and retracting the conjunctiva and muscles, checks the hemorrhage by packing the cavity with hot moist boric acid cotton compresses.

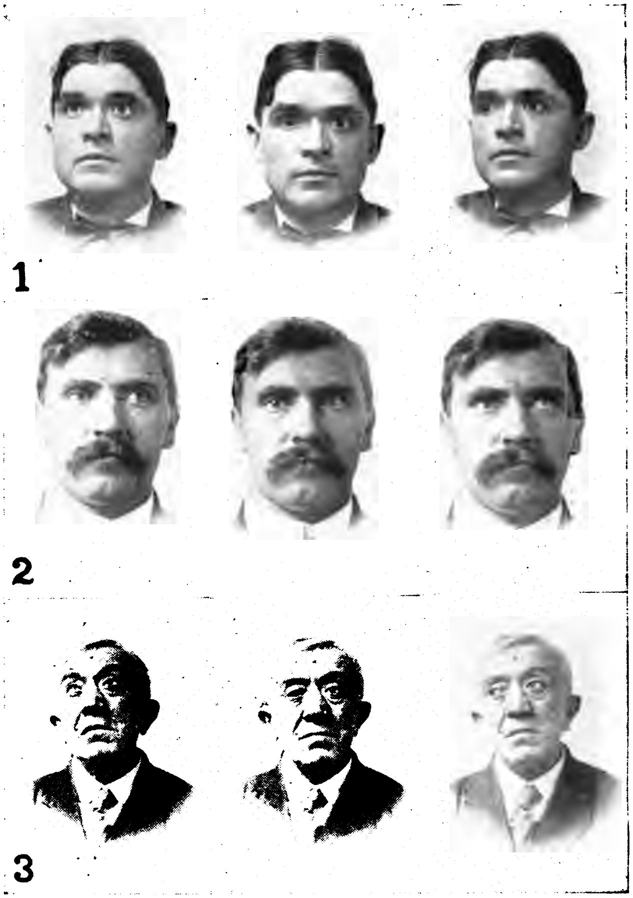

Fig. 5.—In this and Figure 6, cases selected at random from the last twenty-six illustrate the adaptability of the operation to all conditions requiring enucleation. 1. Left eye, exudative iridocyclitis with increased tension; eye blind and painful. 2. Right eye, phthisis bulbi following penetrating injury through cornea and ciliary region. 3. Left eye, absolute glaucoma.

The abdomen, which has been prepared previously, is now bared and a vertical incision is made in the median line beginning just below the umbilicus and carried downward about 2 inches, cutting through to the aponeurosis of the rectus abdominalis. At the bottom of this incision a similar one is made at right angles and a flap dissected which contains the full thickness of the abdominal fat. A piece of fat which should include the subcutaneous tissue and which should be *about the size of the enucleated eye* is then

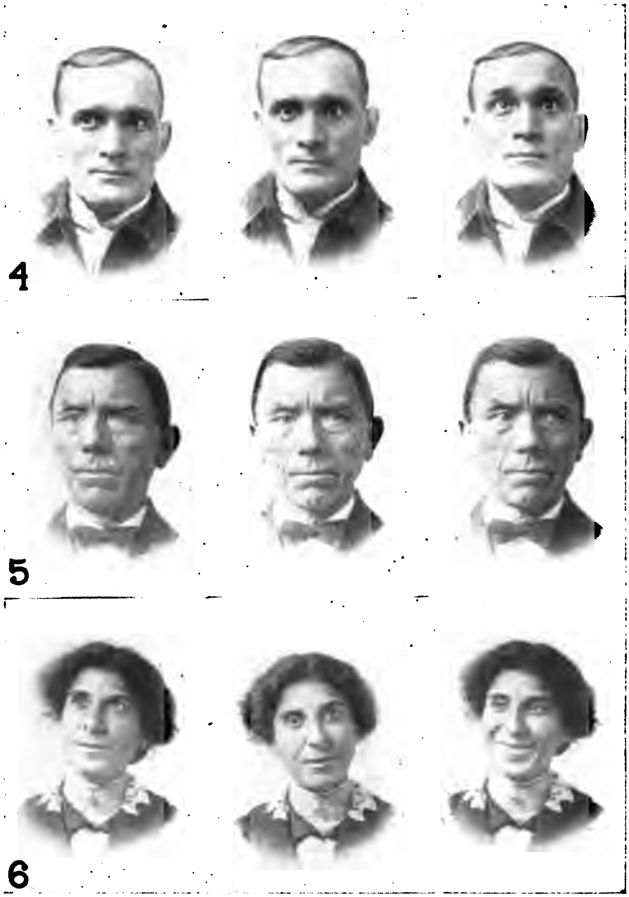

Fig. 6.—4. Left eye, melanosarcoma of choroid. 5. Right eye, incised wound through upper lid, cornea and sclera; lid sutured and eye enucleated with transplantation of fat at the same time; mobility good, but upper lid thickened and stiff from scar. 6. Right eye, large opaque staphyloma of cornea.

excised. The abdominal wound is turned over to a second assistant who checks hemorrhage and sutures the integument. The excised mass of fat is then introduced into the orbit (hemorrhage from which is usually checked by this time) *with the subcutaneous tissue upward,* so as to provide for union between it and the edges of the severed ocular muscles and conjunctiva. The catgut sutures are then tied together, traction is made on them and a purse-string suture of No. 0 twenty-day chromic catgut is passed through the conjunctiva and firmly tied (Figs. 2 and 3). Figure 4 shows the appearance of the stump three weeks later, after absorption of the sutures. I allow wearing of the prosthesis as soon as this occurs.

There is no reaction following the operation; the lids do not become swollen or ecchymosed, the only precaution which I insist on being to fill the eye with my modification of White's ointment[1] after the operation and daily thereafter for a week. The time required in the operation is usually about thirty minutes.

There will result a certain amount of absorption of the fat, varying from a third to a quarter of its bulk, but this usually ceases within six weeks after the operation, leaving a soft, mobile cushion on which the ordinary Snellen eye rests comfortably and moves with the sound eye.

Westinghouse Building.

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[1] As I stated in an article on "The Treatment of Ulcer of the Cornea," (Pennsylvania Med. Jour., June, 1907), I have modified and I believe improved the formula originally suggested by Dr. White of Richmond by adding a small amount of lanolin in which to dissolve and work up the mercuric chlorid and sodium chlorid, as follows:

| | |
|---|---|
| Hydrargyri chloridi corrosivi | gr. i |
| Sodii chloridi | gr. v |
| Adeps lanae hydrosi | q. s. |
| Petrolati | ℥ vi |

The sodium chlorid and mercuric chlorid are dissolved and rubbed up in about 1 dram of lanolin; the petrolatum is boiled for five minutes, the impregnated lanolin added and, after boiling for five minutes longer, the liquid ointment is poured into jars. After it has cooled it will be found to be of a proper consistency to introduce under the lids, becoming slowly melted by the heat of the body and permeating to all parts of the conjunctival sac.

## MEMORANDA

# MEMORANDA

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617+

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